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## Table of Contents.





## E. H. Embley Memorial Lecture.[1]

### IF EMBLEY WERE TO RETURN.

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EDWARD HENRY EMBLEY, whom we honour this evening, died in 1924. He was a modest and retiring man, whose services to anæsthesia were not adequately appreciated in his own country during his lifetime. He gained abroad the recognition which he missed at home, and his name is still honoured in America. He is, indeed, possibly the only Australian anæsthetist who has won international recognition. This fact reflects no credit upon the progress of medical science in Australia, but is not difficult of explanation.

In anæsthesia, as in other branches of medicine, the easy discoveries have by now been made. It has become difficult for a brilliant amateur of research, such as Embley was, to achieve important results in clinical practice and with improvised laboratory equipment. Research has become specialized, demanding close collaboration between the manufacturing chemist, the physiologist, the experimental pharmacologist and the clinical anæsthetist. It has therefore centred in great university hospitals, as at Oxford, Madison, New York and elsewhere. It demands not only trained men, but also elaborate equipment and generous endowments. Lacking these, Australia can scarcely hope to produce work of the first order. Her practising anæsthetists, at their best, measure up to overseas standards, but their working conditions are unfavourable to originative effort. They are, broadly speaking, not so much originators as exponents and developers of the ideas of others. Hence it is that no major advance in anæsthesia has come out of Australia since Embley's day, nor is any likely until anæsthesia gains academic recognition and facilities for whole-time research. The application of teamwork to research in anæsthesia originated in America: in the British Commonwealth, a fine beginning has been made with the Nuffield Department of Anæsthetics at Oxford, but in Australia there exists only a vision for the future.

Embley retired from practice in 1920, a quarter of a century ago. It will be profitable this evening to attempt to picture what he would find were it possible for him to return to life; what progress he would recognize in the speciality which he loved and served so well.

#### The Organization of Anæsthetists.

Like his contemporaries, Embley combined anæsthesia with other forms of practice. Specialist anæsthetists are still few in this country; of the members of our Society of Anæsthetists, perhaps one in five confines his work solely to anæsthesia. The reason for this is economic; it is not easy to gain a livelihood in Australia by the exclusive practice of anæsthesia.

The Australian Society of Anæsthetists was founded in 1934, ten years after Embley's death. It has been handicapped by the economic conditions of practice, which tend to limit the anæsthetist's interests to his daily concerns. Travel and the exchange of ideas, the life-blood of any scientific society, are difficult in these circumstances. Australia lacks also that class of salaried teachers which, in America, provides the main support of organized anæsthesia and its great reservoir of original ideas. Embley, were he to return, would probably feel that organized anæsthesia in Australia has achieved little more than a gesture towards the future.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on April 4, 1945.

### The Teaching of Anæsthesia.

Anæsthesia, the Cinderella of the medical specialities, has received but small consideration in the curriculum. In Melbourne, for example, the course has reverted during the war to much what it was in Embley's day, namely, four lectures and six practical administrations. It is difficult to justify this logically when twenty-six lectures are devoted to a reading course in German. A recent graduate, on entering upon a house appointment or on joining the forces, will certainly be required to give anæsthetics. For this work he is ill-equipped: his training, indeed, is much less than that of the dental student, who in Melbourne must attend sixteen lectures and perform forty administrations. In consequence, the medical graduate must pick up a knowledge of anæsthesia as best he can after graduation, without systematic instruction and all too often by the process of learning from his mistakes.

If undergraduate teaching is unsatisfactory, post-graduate teaching hardly exists at all. An occasional course of instruction is arranged by the New South Wales Post-Graduate Committee in Medicine, and a Diploma in Anæsthesia has been recently initiated in the University of Sydney. It is unfortunate that this forward step could not have been taken in collaboration with the other Australian universities and with the Royal Australasian Colleges of Physicians and Surgeons. Such a diploma might well be a prerequisite to a teaching post in anæsthesia in every hospital.

In Embley's time, the field of anæsthesia was limited and many men entered it because they were unambitious or physically handicapped. The speciality has become a wide and a strenuous one, giving ample scope to men of keen mind. If it is to flourish, it must have constant recruitment. Promising young graduates will enter it, however, only if it is made attractive. Security is essential, whether it comes from the steady support of surgeons or from the creation of salaried appointments. The anæsthetist, unlike other medical specialists, does not look directly to the general public; he depends upon the support of his surgical colleagues. The demands of his private practice are therefore apt to conflict with those of his hospital work. Surgeons in public hospitals hence receive less than their due share of specialist anæsthetic assistance, the burden upon the resident staff is increased, and the opportunities for training students are curtailed. The time is approaching when our public hospitals may have to consider the appointment of salaried anæsthetists, as in the United States. Men so employed would have every incentive to make their speciality a life's calling and to equip themselves thoroughly for it. To secure the right type of applicant, however, the service would need to be attractive and to offer facilities for post-graduate study. The need for systematic post-graduate instruction is an urgent one in nearly all the capital cities of Australia.

From the unimpressive picture of the present-day organization and teaching of anæsthesia in Australia, we turn to a more gratifying one, namely, the technical progress which has been made in the speciality since Embley's day.

### Anoxia.

Anoxia, hypoxia or oxygen lack are terms used to indicate an intake of oxygen insufficient for the metabolic needs of the patient. The term "anoxia" is a fairly recent one, being used by Hewer[1] only in the 1939 edition of his book. In Embley's time, the term "anoxæmia", or deficiency of oxygen in the circulating blood, was used with similar connotations. It is now realized that grave anoxia may exist without anoxæmia, for example, where the circulation time is prolonged because of myocardial insufficiency, or where the ability of the tissue cells to make use of the oxygen in the blood is inhibited by toxins, amongst which must be included the anæsthetics. The manner of action of the anæsthetics is obscure and complex, and cannot be discussed in the limits of this paper. Briefly, the theory of widest current acceptance is that which supposes the anæsthetics to act by inhibiting the intracellular respiratory hormones. The result is interference with the internal respiration of the tissues, and especially with that of the central nervous tissues. By this conception, very deep anæsthesia must necessarily imply tissue anoxia, and anoxia of any origin must add greatly to the hazards of anæsthesia. Hence, the current literature of anæsthesia places more stress upon the avoidance of oxygen lack than was the case in Embley's day.

The general interest in anoxia has led to a revaluation of cyanosis. The older anæsthetists, using volatile anæsthetics, always regarded it as a danger signal. McKesson[2] taught that it might be ignored in the case of nitrous oxide, provided that no ocular, respiratory or circulatory signs of oxygen lack were present. It is now agreed that

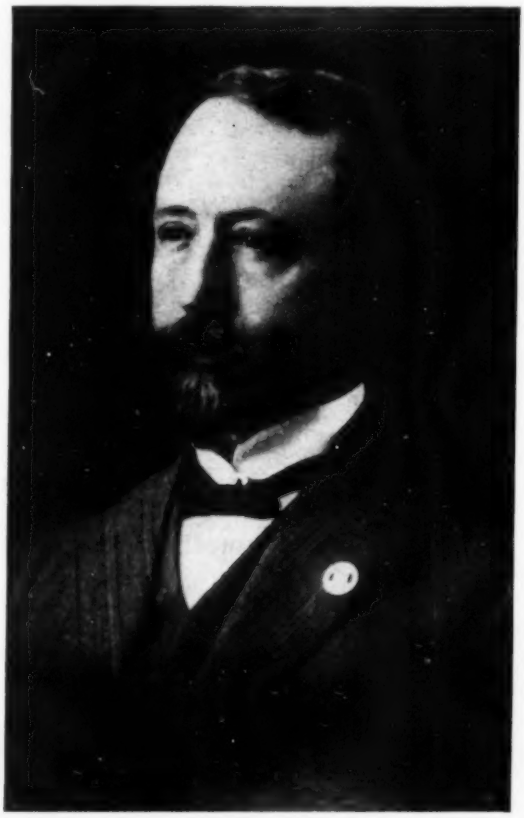

FIGURE I.

E. H. Embley, 1861-1924. (Brooklyn Studios.) By courtesy of the Victorian Branch of the British Medical Association.

cyanosis always indicates oxygen lack, except in known and proven plethorics, and is therefore to be strictly avoided. The reasons for this conclusion are well set out by Macintosh and Bannister.[3] McKesson and his followers were really reinforcing the weak anæsthetic action of nitrous oxide by some degree of anoxia. This may be done with comparative impunity under nitrous oxide, which is non-toxic, but it would lead straight to disaster with ethylene or cyclopropane.

The signs of oxygen lack are presented graphically by Adriani.[4] Respiration may be initially accelerated owing to the influence of the carotid sinus, but soon becomes slow, shallow, irregular or periodic. The pulse rate rises; the blood pressure, after transient elevation, falls progressively. Muscular rigidity appears and leads to eccentric fixation of the eyeballs; later, asphyxial twitchings may become apparent. It will be seen from this description

that the signs of anæsthesia which were recognized in Embley's day, and especially the signs of gas anæsthesia, were in part the signs of anoxia. The confusion is explicable from the probable manner in which anæsthetics act. The true signs were first separated by Guedel,[5] whose classification has gained world-wide acceptance. Its most detailed revision is that of Gillespie.[6]

Anoxia is classified by the physiologists into the anoxic, anæmic, stagnant and histotoxic types, these being described in all the text-books. Their causes are manifold, ranging from an ill-regulated anæsthetic mixture to hæmorrhage or myocardial insufficiency. The result in

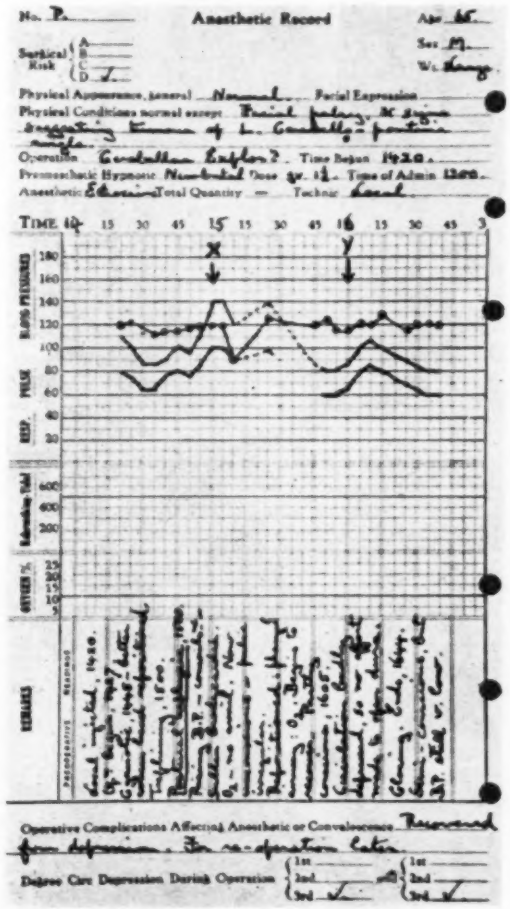


FIGURE II.

Effect of asphyxiation. The patient was undergoing a cerebral operation under local analgesia. Owing to postural asphyxiation, temporary elevation of the blood pressure occurred (X) with irregularity of the pulse. As the asphyxiation could not be relieved, the circulation became so depressed (Y) that the operation had to be abandoned. (From a paper by the author in *The Australian and New Zealand Journal of Surgery*, 1937, Volume VII, page 134.)

either case is a vicious circle; the less well-oxygenated the cerebral centres, the less adequate are the intake of oxygen and the circulation of oxygenated blood. As a side-effect to this vicious circle, there is progressive deterioration in the renal and hepatic functions. The proximate remedy is oxygen therapy, which is not always employed in a physiological manner. It should be commenced early, before anoxia has made the patient restless and non-cooperative. The concentration of oxygen should be initially the highest that is mechanically possible.[7][8]

### Carbon Dioxide.

The use of carbon dioxide as a respiratory stimulant in anæsthesia is later than Embley's time, entering general use about 1925. It enjoyed a vogue which was often uncritical and which has only lately become more reasoned.

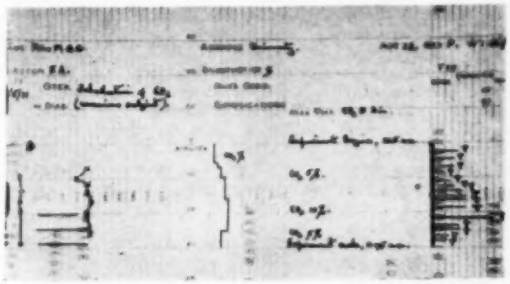

FIGURE III.

Effect of inhalation of carbon dioxide. A conscious subject inhaled varying percentages of the gas in air, a record being made meanwhile with a McKesson Recording "Nargraf" apparatus. There was no material change in the rate of pulse and respiration, but the systolic blood pressure rose and the tidal volume of respiration increased to nine hundred cubic centimetres. This shows how easily the respiratory centre can be overstimulated, and eventually fatigued, by carbon dioxide. (From a chart used by the author in "Practical Anæsthesia", 1932.)

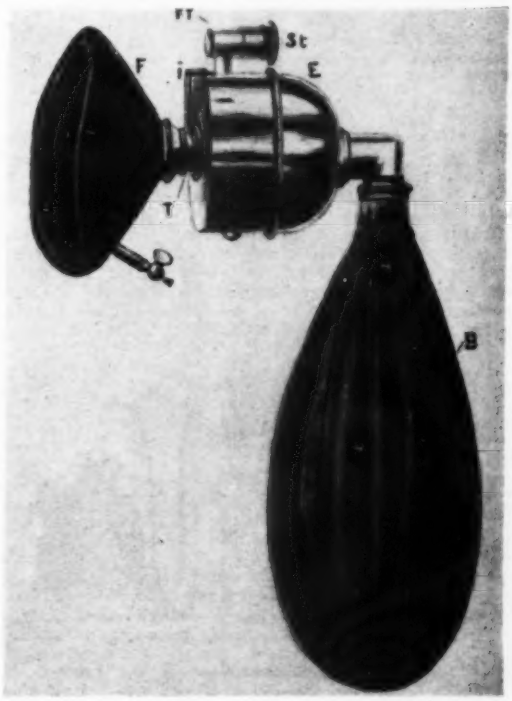


FIGURE IV.

Clover's ether inhaler, 1876. (From Hewitt, 1893.) An example of this apparatus, used by Embley in his earlier years of practice, is preserved in the museum of the Australian Society of Anæsthetists.

The relative shares of carbon dioxide and oxygen in sustaining the respiration are well expressed by Henderson.[9] Briefly, oxygen lack is the stimulus to respiration and carbon dioxide is a potent stimulant to the respiratory centre. Its excessive administration can over-stimulate and fatigue that centre, as well as disturb the acid-base equilibrium of the blood.

The anæsthetics, the narcotics and oxygen lack act in a similar way upon the respiratory centre, in that they all raise the threshold of its sensitivity to carbon dioxide. This fact explains the slow and periodic respiration seen in anæsthetic overdosage or in gross anoxia; the respiratory centre must await the accumulation of the carbon dioxide in the blood to the new threshold value before a respiratory cycle can be initiated. The patient's urgent need is for oxygen. The giving of carbon dioxide to stimulate the intake of this oxygen is not without hazard, for the carbon dioxide tension of the blood is already high and further administration may well over-stimulate the respiratory centre. The carbon dioxide should be withdrawn, therefore, as soon as the threshold value has been reached, that is, as soon as the respiration has recovered its normal amplitude. Further administration may do irreparable harm.

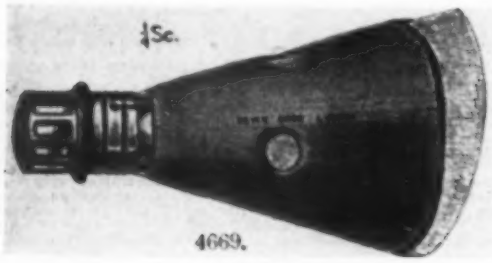


FIGURE V.

The Hyderabad cone, 1890 (from an old catalogue). The cone was recommended by the Hyderabad Chloroform Commission, and it was well-founded disbelief in the findings of the commission which started Embley upon his researches.

Reduction in the carbon dioxide tension of the blood is called acapnia, and may result in the cessation of respiration until the normal threshold is again attained. It is almost impossible to produce acapnia by the usual anæsthetic methods; enough "dead space" exists within the apparatus to supply the minute quantity of carbon dioxide necessary for continued respiration. A possible exception exists in endopharyngeal or endotracheal insufflational anæsthesia, if the delivery is greatly in excess of the minute volume of respiration; even here, the apnœa will cease shortly after the excessive delivery is curtailed.

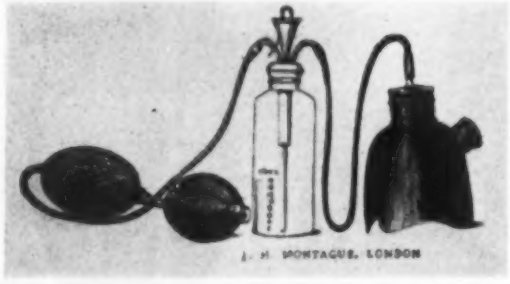


FIGURE VI.

Junker's chloroform inhaler, 1867. (From Ross and Fairlie, 1929.) An apparatus very popular in Embley's day.

An excessive tension of carbon dioxide in the blood results in hyperpnœa and elevation of the systolic blood pressure. It is one of the commonest faults in anæsthetic technique, resulting from such causes as over-large masks, excessive draping with towels, undue "dead space" in the apparatus or excessive rebreathing. The patient, being continually stimulated with carbon dioxide, may appear to be very well "on the table", only to become gravely depressed when this stimulus is withdrawn at the end of the administration.

Excess of carbon dioxode is not synonymous with oxygen lack, although the two conditions may coexist, as in respiratory obstruction. Uncomplicated carbon dioxide excess does not, of itself, cause cyanosis; the latter appears only when the respiratory centre is failing because of over-stimulation and the picture is becoming complicated by oxygen lack.


FIGURE VII.

Shipway's ether vapour apparatus, 1915. (From Ross, 1919.) Used in the British army during the war of 1914-1918. Of wide employment before the general use of endotracheal methods.


FIGURE VIII.

The Oxford vaporizer, 1941. (From Macintosh and Bannister, 1943.) A refined example of the modern wide-bored inhalational vaporizer for use with volatile anæsthetics.

### The Volatile Anæsthetics.

In 1920, the anæsthetics in common use in Australia were ether, ethyl chloride and chloroform. Nitrous oxide, whilst it had given good service in the first German war, had not attained to popularity. It was left mainly to the dentists, who gave it as a "single-dose" anæsthetic by methods frankly asphyxial. Ethylene and cyclopropane were still in the womb of the future.

Ether was then, as now, the "standard" anæsthetic. Embley began his career with the Clover's inhaler, which dominated British practice for a generation and displayed to the full the ill-effects of high concentration, restricted air passages, depletion of oxygen and accumulation of carbon dioxide. The "open" method, introduced in 1895, replaced the closed inhaler during the first decade of the twentieth century. Its general adoption in Australia was largely due to the advocacy of R. W. Hornabrook and of Embley. Ether still holds pride of place with us, although

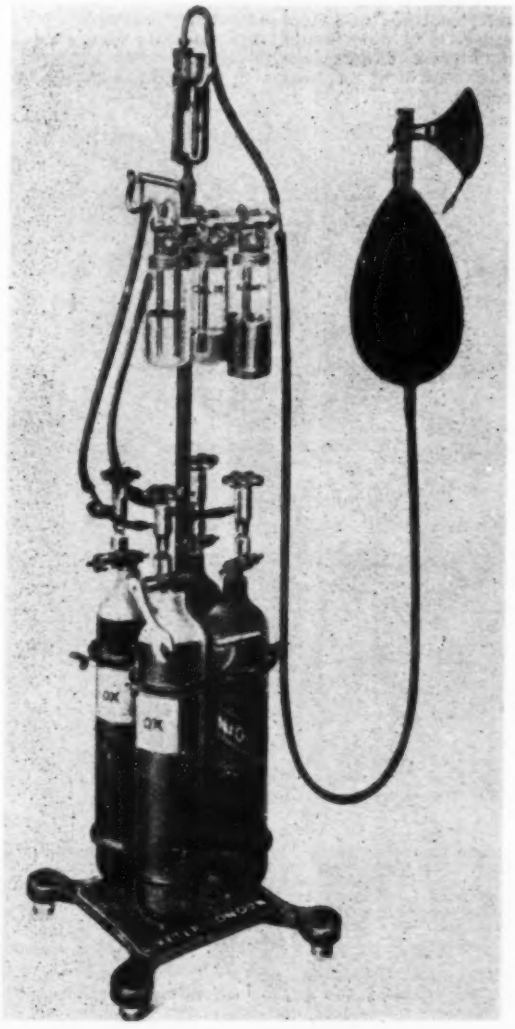

FIGURE IX.

Boyle's gas anæsthesia apparatus, 1915 (from a catalogue). Used in the British army during the war of 1914-1918. An important landmark in the evolution of anæsthesia apparatus of the water flowmeter type.

our methods of giving it have improved. The "open" method has become more truly open, with less draping of the mask and consequently with better oxygenation and less accumulation of carbon dioxide. The modern tendency is to employ inhalational vaporizers of the Oxford or similarly wide-bored type, which supply a vapour of uniform and controllable strength which is not chilled like that beneath an "open" mask. Despite its pungency and its nauseating effects, ether is an excellent anæsthetic. It is simple of administration, has a wide margin of safety, produces satisfactory muscular relaxation, and (with modern methods of administration) is probably less irritating to the lungs than was formerly believed. It is admirably suited to the needs of the occasional anæsthetist and is the yardstick by which other anæsthetics are gauged.

Embley, were he to return, would find chloroform under a cloud. Its dangers, to the elucidation of which he contributed so materially, are now well-recognized. Its scope, already narrowed by the general use of the anæsthetic gases, has been further restricted by the introduction of trichlorethylene.[10][11] This drug, which is non-inflammable and non-irritating to the lungs, seems able to do nearly all that chloroforom once did, and to do it with greater, although by no means with absolute, safety. Embley's explanation of primary cardiac arrest under chloroform[12]

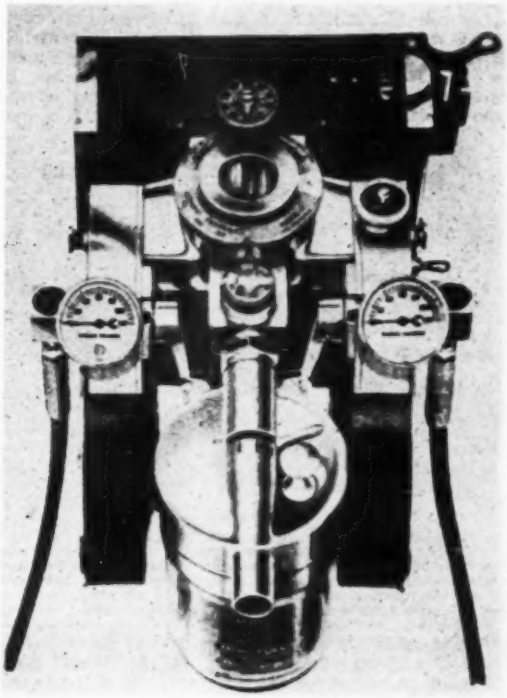

FIGURE X.

McKesson's gas anæsthesia apparatus, 1929. (From McKesson, 1935.) A refined example of apparatus for administration by the intermittent flow method.

is not now the accepted one. He attributed it to irritation of the vagal centre, so that a trivial surgical stimulus might overflow into the irritable vagus and initiate arrest of the heart beat. This explanation, whilst perhaps sufficing in those fatalities which occur on surgical stimulation under light anæsthesia with chloroform, does not account for those in which no stimulus has been applied. During Embley's lifetime, Goodman Levy[13] had succeeded in producing ventricular fibrillation at will in cats lightly anæsthetized with chloroform. Subsequent electrocardiographic studies[14] have shown chloroform to act upon the conductional tissue of the heart, displacing the pacemaker downwards from the sino-atrial node towards the atrio-ventricular. Nodal rhythm is thus produced, with ventricular extrasystoles and, in some instances, short periods of ventricular tachycardia. This is very likely to result in ventricular fibrillation, the more so because chloroform is said to shorten the refractory period of the cardiac muscle. If the fibrillation is of more than momentary duration, the resulting ischæmia

will destroy the contractility of the cardiac muscle, so that the circulatory arrest will become permanent. Further, the fall in the blood pressure to zero will produce complete anoxia of the vital medullary centres.

Ventricular fibrillation can occur, and has occurred, under any anæsthetic, especially on interference with a "reflexigenous" area at a light plane of anæsthesia. Its special frequency under chloroform seems to be due, not to irritability of the vagus, as Embley believed, but rather to the displacement of the pacemaker and production of ventricular tachycardia. It was known in Embley's day that sudden death under chloroform was especially likely if adrenaline was employed by the surgeon. The reason for this has been only recently elucidated by Meek and his co-workers,[15] who found chloroform able to sensitize the conductional tissue of the heart to the action of adrenaline, whether introduced by the surgeon or secreted by the patient under the influence of apprehension. The effect of the adrenaline is to aid the downwards displacement of the pacemaker which predisposes to ventricular fibrillation. Embley's views, then, may not be our present ones, but they stimulated world-wide interest when they were announced and they led the way to further advances in knowledge.

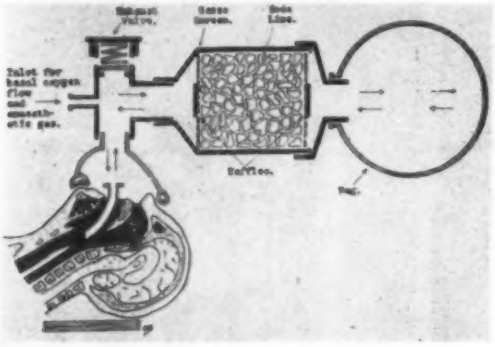

FIGURE XI.

Diagram to illustrate the principle of anæsthesia by the "to and fro" method of carbon dioxide absorption. The anæsthetic gas, supplied from a standard anæsthesia apparatus, is rebreathed to-and-fro through a canister containing soda lime, which removes the carbon dioxide. Oxygen is supplied at a rate dictated by the patient's metabolic needs.

The pharmacology of ethyl chloride was investigated by Embley,[16] and its administration by the "open" method owed much to his advocacy. The agent is still in general use, although its potency and potential toxicity are well recognized. It is therefore used less as an anæsthetic *per se* than as a prelude to ether, being given in merely amnesic dosage.

### Gaseous Anæsthetics.

It is improbable that Embley had any great experience of gas anæsthesia, for the method was late in reaching Australia. In 1920, the only gas in use was nitrous oxide. The method of administration usual in England or the eastern United States was that of continuous flow, with the use of the water-flowmeter apparatus of Gwathmey, Foregger or Boyle. McKesson's ingenious development of an intermittent flow method[17] had not, at that date, extended far beyond the middle western States of America.

McKesson's technical dexterity led him to advocate the dangerous practice of "secondary saturation", in which the anæsthetic effects of nitrous oxide were deliberately reinforced by oxygen lack. The method proved, in the hands of some of his followers, a prolific source of anoxic cerebral sequelæ, and little is now heard of it. McKesson was also the most successful exponent of the administration of gaseous anæsthetics under positive pressure, which was regarded in the 'twenties as the last word in technique. Undoubtedly, the ability to sustain flagging respiration by forcing a respirable mixture past an obstruction, or by encouraging the diffusion of oxygen inwards through the pulmonary endothelium, was and is most helpful. It is now realized, however, that the routine use of positive pressure is actually disadvantageous. It obliges the patient to exhale against a gaseous pressure, thus causing respiratory fatigue. It also prevents the lungs from deflating normally during exhalation; the respiration is therefore not fully effective, because a portion of the gases in the lungs remains unchanged. The intake of oxygen and the elimination of carbon dioxide are therefore less adequate than in normal breathing at atmospheric pressure. Rather than to continue prolonged administration under positive pressure, it is now thought preferable to secure patency of the airways by laryngeal intubation, or to take over the manual control of respiration in the manner shortly to be described.

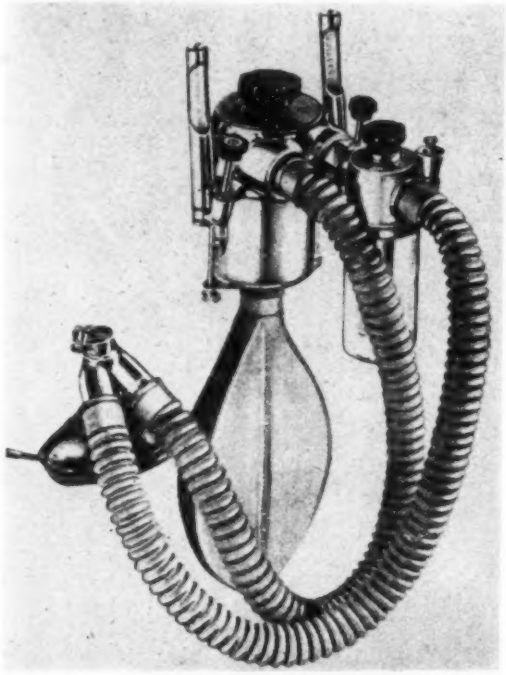

FIGURE XII.

A representative absorber head, for use in the closed-circle method of carbon dioxide absorption. It is shown detached from the anæsthesia apparatus, and comprises mask, corrugated hoses for inhalation and exhalation, non-return valves ensuring uni-directional movement of the gases, a canister containing soda lime, and dry flowmeters for oxygen and cyclopropane.

The outstanding event of the 'thirties was the world-wide adoption of the carbon dioxide absorption method, which was introduced by Waters[18] so early as 1923. Briefly, the method consists in the complete rebreathing of the same charge of anæsthetic mixture. The carbon dioxide is removed from it by filtration through soda lime; oxygen is added at a rate dictated by the patient's metabolic requirements, and the anæsthetic gas is inhaled and exhaled unchanged. This technique produced a veritable revolution in the attitude of anæsthetists, not only towards gas anæsthesia, but towards inhalational anæsthesia in general. Firstly, the atmosphere in an absorption system is always warm and moist, so that the patient is found to sweat less, to lose less bodily heat and to retain better circulatory tone. Secondly, the respiration assumes a quietude found in no other form of anæsthesia. There are two reasons for this, one being the absence of respiratory stimulation by carbon dioxide, and the other being the freedom from the respiratory resistance which is

conferred by air passages seven-eighths of an inch in calibre and by effortless valves. Anæsthetists all the world over were hence led to view their apparatus with more critical eyes, whether it were intended for use with gaseous

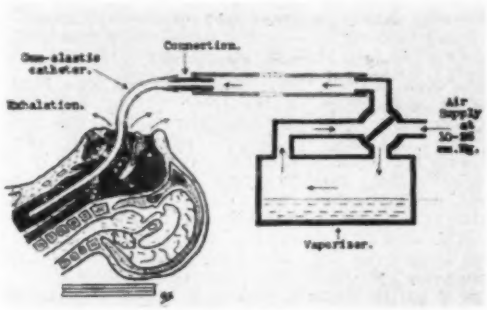


FIGURE XIII.

Diagram to illustrate the insufflational method of endotracheal anæsthesia. The anæsthetic mixture, supplied under positive pressure from a vaporizer, is insufflated through a small-bored catheter into the patient's trachea. The return flow of air has to find its own way out between the catheter and the laryngeal walls, theoretically blowing back foreign matter from the trachea in its passage. Packing of the pharynx is inapplicable, lest the return flow be obstructed and surgical emphysema result. This was the method of endotracheal anæsthesia used in Embley's day.

or volatile anæsthetics. Channels were widened, valves were made effortless and "dead space" was eliminated. Respiration has assumed a new quietude, which would be gratifying to Embley the physiologist, if he could see it.

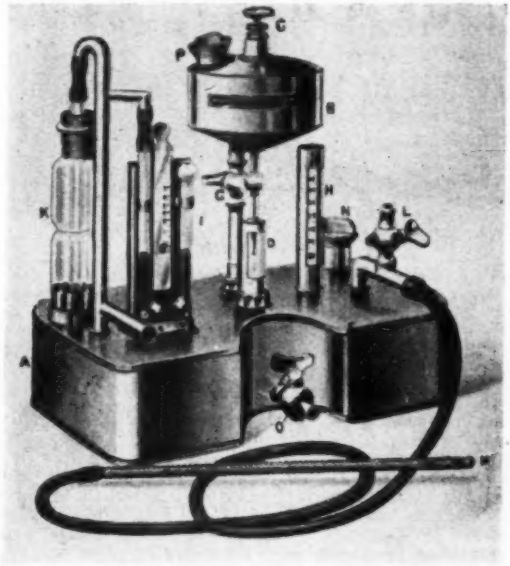

FIGURE XIV.

Shipway's "drip-feed" endotracheal ether apparatus, 1916. (From Ross, 1919.) A representative apparatus of the type used for endotracheal insufflation in Embley's time. Note the small-bored, semi-rigid catheter.

An important extension of the absorption technique is "controlled respiration", which was introduced by Guedel. Without it, modern thoracic surgery would hardly be possible. The method consists in first raising the carbon dioxide threshold of the respiratory centre by the use of heavy premedication or of a potent anæsthetic such as ether or cyclopropane. If, now, the carbon dioxide tension of the blood is kept at a normal level by ventilating the lungs through soda lime, the centre will be deprived of its stimulus and spontaneous respiration will cease. In practice, the ventilation is secured by manual compression of the breathing bag during inhalation. Once apnœa supervenes, it may be prolonged at will by rhythmical compression of the bag, which also serves to oxygenate the lungs. The quiet respiratory movement so produced aids in the exposure of deeply situated thoracic or abdominal structures. Of still greater importance is the fact that manually controlled respiration is more efficient than that which a patient with an open pneumothorax can manage for himself, owing to the devastating effects of mediastinal "flap" and paradoxical respiration. The application of "controlled respiration" to thoracic surgery is described in a masterly paper by Nosworthy.[19]

Nitrous oxide remains a popular anæsthetic, but its low potency is now recognized. No attempt should be made to reinforce it by oxygen lack; the supply of oxygen must be abundant, and relaxation be assured if necessary by a small supplement of ether or other volatile agent. This course is far safer than is any attempt to force the action of nitrous oxide in presence of anoxia, whilst there is no evidence that it adds to the incidence of post-anæsthetic vomiting or respiratory sequelæ. Anoxia increases both, and that is the least of its possible harmful effects.

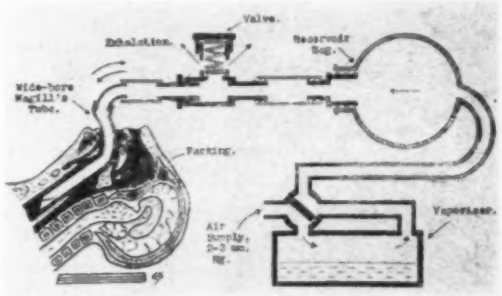


FIGURE XV.

Diagram to illustrate the "semi-closed" method of endotracheal inhalational anæsthesia, introduced by Magill in 1920. A wide-bored rubber tube is used, through which the patient inhales anæsthetic mixture delivered at low pressure into a bag. Exhalation takes place through a valve of bore equal to that of the endotracheal tube. Effective packing of the pharynx, or occlusion of the trachea around the tube by the use of an inflatable cuff, now becomes possible. For gas anæsthesia, the vaporizer may be replaced by a standard anæsthesia apparatus of either the continuous-flow or intermittent-flow type.

Ethylene has been, in the main, supplanted by cyclopropane. This, the most potent and efficient of the present anæsthetic gases, has one drawback. In high concentrations it produces disturbances in the conductional tissue of the heart not unlike those which occur under chloroform. Prolonged deep anæsthesia with cyclopropane is therefore of questionable wisdom. The gas may be given with a high percentage of oxygen, and is therefore of the greatest value in cases of actual or potential anoxia of the anoxic type, for example, in respiratory obstruction or in thoracic surgery.

### Endotracheal Anæsthesia.

In Embley's time, endotracheal anæsthesia was conducted by the method of insufflation under positive pressure through a small gum-elastic catheter. In 1920, Magill[20] introduced the present inhalational method, which employs the largest rubber tube which the larynx will comfortably accommodate. An airway is thus assured for exhalation as well as for inhalation; the respiration is quiet because it does not have to take place against positive pressure, and the pharynx can be securely packed to exclude foreign matter from the bronchial tree.[21] A small suction catheter may be passed down the main tube, permitting of intermittent drainage of the trachea without interruption of the anæsthesia.

Magill's original method was a "semi-closed" one, the anæsthetic mixture being delivered at low pressure into a reservoir bag whence it was inhaled by the patient. Exhalation took place through a valve interposed between endotracheal tube and bag. The modern practice is to connect the valve directly to the wide-bore delivery tube of an inhalational vaporizer such as the Oxford; no pumping action is now required, the patient inhaling air freely across the surface of the anæsthetic. In gas anæsthesia, the endotracheal tube may be connected to a closed system which includes soda lime. Respiration in this event is as quiet as it well can be, for the patency of the airways is assured, the atmosphere is warm and moist and there is no stimulation by carbon dioxide.

The realization that no patient need ever die from asphyxiation if an endotracheal tube is available is older than Magill's technique. The latter marked, however, a revolutionary advance. It pointed the way to that elimination of respiratory resistance which characterizes modern anæsthetic technique. It gave new security to the bronchial tree, and permitted of effective tracheal drainage. When combined with carbon dioxide absorption and with "controlled respiration", it placed the anæsthetist in full control of the respiratory function. If, in the future, a like degree of control can be exercised over the circulatory function, the day of really safe anæsthesia will have dawned.

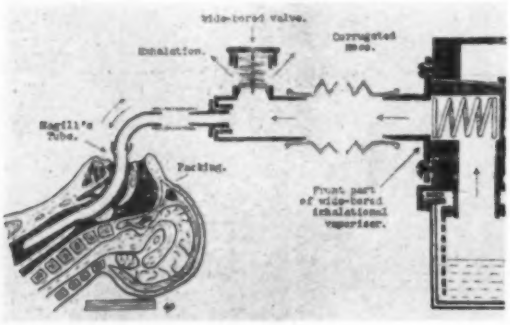


FIGURE XVI.

Diagram to illustrate the inhalational method of endotracheal anæsthesia. A standard Magill's tube is used, the pharynx being securely packed. The patient now inhales the anæsthetic mixture through a wide-bored hose attached to an inhalational vaporizer of wide bore. Exhalation takes place through a valve, also of wide bore, so that restriction is confined to the endotracheal tube and its immediate connexions. For gas anæsthesia by the method of carbon dioxide absorption the endotracheal tube is connected to either the Waters canister shown in Figure XII or the closed circle absorber shown in Figure XIII.

### Intravenous Anæsthesia.

The story of intravenous anæsthesia is largely the story of the short-acting derivatives of barbituric acid. Of these, the first was introduced in 1920, the year of Embley's retirement; its name was somnifaine. Ever shorter-acting and less toxic barbiturates have been since evolved, culminating in "Evipan Sodium" in 1932 and "Pentothal Sodium" in 1934.[32] At the present time, intravenous anæsthesia is greatly in vogue. It is popular with the lay public, and there can be no doubt as to its convenience, portability or efficiency. The only rational method of administration is the fractional, in which doses of one-half or one cubic centimetre of the solution are given as and when necessary for the maintenance of anæsthesia. These increments may be conveniently added to an intravenous drip infusion of saline solution. There is possibly no operation of surgery which has not been performed under this form of anæsthesia, but its comparative safety still remains to be evaluated.

The barbiturates are powerful respiratory depressants, and oxygen should be always at hand when they are to be administered. They are contraindicated by anoxia of every type, whether obstructive, hæmorrhagic or cardiac in origin. Further, they increase the irritability of the respiratory tract, so that grave laryngospasm may follow their use in cases in which blood, pus or morbid secretions may gain access to the hypopharynx or glottis. According to Weese[33] they must be used with special caution in cases of phlegmon of the neck, lest the surgical incision provoke a carotid sinus reflex and respiratory arrest.

### Hepatic Function.

Much work has been done since Embley's time upon the effects of anæsthetics on the liver. Using dye tests of hepatic function, Bourne[34] has shown that the gaseous anæsthetics produce no harmful effects unless administered with a deficiency of oxygen, that the effects of ether are definite but transitory, and that chloroform causes marked damage which may range from prolonged dysfunction at the best to central lobular necrosis at the worst. He and his co-workers have also demonstrated that the best way to protect the liver is to avoid anoxia and to build up an adequate pre-anæsthetic reserve of glycogen. The giving of barley sugar or dextrose in the evening before operation has therefore become standard practice, and has been reflected in a gratifying decline in the incidence of post-anæsthetic vomiting.

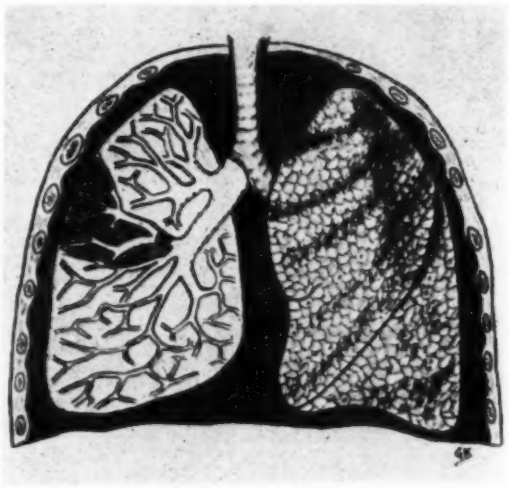

FIGURE XVII.

Diagram to show the production of pulmonary atelectasis. The right lung is drawn in section, showing the bronchial tree. Occlusion of a branch bronchus by a plug of mucus is followed by atelectatic collapse of the area of lung tissue behind the obstruction.

### Respiratory Sequelæ.

The incidence of post-anæsthetic bronchitis or bronchopneumonia was apparently high in Embley's day. The share of pulmonary atelectasis in its causation was recognized by about 1930, but it seems in retrospect that undue prophylactic value was attributed to inhalations of carbon dioxide. The more modern view of the subject is well presented by Waters.[35][36] At the end of anæsthesia, undue accumulation of mucus must be aspirated from the hypopharynx and trachea, the latter being intubated if necessary. As soon as possible after his awakening, the patient should be encouraged to sit up and to move about in bed. A "stir-up" *régime* of active coughing and deep breathing should be enforced once in every waking hour. Inhalations of carbon dioxide will have subsidiary value, since they promote expansion of the lungs. If atelectasis occurs despite these measures, it is essential that it be overcome before the collapsed pulmonary tissue undergoes autoinfection. Bronchoscopy must accordingly be performed, so that the plug of tenacious mucus may be aspirated from the obstructed bronchus. The occurrence of bronchopneumonia is usually the result of auto-infection of an

atelectatic area of lung. The mortality in the past was high, but a more hopeful outlook exists since the introduction of the sulphonamides and of penicillin.

### Spinal Analgesia.

Many of the unfortunate sequelæ of spinal analgesia in Embley's day are now recognized as having been due to irritating and ill-devised solutions. The present conception of the subject dates from 1930, when Howard Jones[27] introduced the hypobaric solution of "Percaine", that is, a solution of specific gravity less than that of the cerebro-spinal fluid. Spinal analgesia is now classified into the "saddle" type, which affects only the sacral nerves, into the "low", which extends to the umbilicus, and the "high". "Saddle" analgesia is most conveniently induced with a hyperbaric solution, that is, with one heavier than the cerebro-spinal fluid. The method is relatively safe, for circulatory depression is uncommon; headache is an occasional sequel, and nervous palsy is not unknown. In "low" analgesia, hypobaric or hyperbaric solutions may be used at the anæsthetist's preference; the hazard of circulatory depression increases with cranial extension of the analgesia. "High" analgesia is best induced with a hypobaric solution. This permits of fairly accurate regulation of the anatomical extent of the analgesia by suitable posturing of the patient, and it also allows the effect to be made unilateral.[28] This, for reasons which will later become apparent, is less likely to depress the circulation than is bilateral analgesia of equal cranial extension. At the same time, "high" spinal analgesia always involves serious risk of circulatory depression, and should be left to those who are prepared to make a special study of it. The same statement applies to the current American practice of continuous spinal analgesia.[29]

The difficulty in spinal analgesia is to predict and control circulatory depression. This, if it occurs, is hazardous both "on the table" and from subsequent reactionary hæmorrhage. Its cause has been much debated. The blocking of the sympathetic vasoconstrictor fibres in the *rami communicantes* and the absorption of the analgesic from the theca into the general circulation probably play a role. The major factor, however, is now thought to be anoxia resulting from intercostal muscular paralysis and from stagnation of blood in the great muscle field of the paretic limbs and trunk. Unilateral analgesia, because it restricts these changes to the minimum, is hence to be preferred wherever applicable. Oxygen therapy is now considered to be even more important than is the giving of analeptics in the treatment of circulatory depression.

### Conclusion.

We have traced, necessarily briefly, the technical advances in anæsthesia since Embley's day. The speciality is a rapidly growing one, and further advances occur almost monthly. Recent progress has been in the direction of avoiding oxygen lack and respiratory resistance, of more clearly appreciating the role of carbon dioxide, of giving more consideration to the renal and hepatic functions, and of understanding more fully the ætiology of respiratory sequelæ. An anæsthetic administration is becoming, to an increasing degree, a demonstration in applied physiology. In the future training of anæsthetists the physiological and pharmacological note will necessarily predominate. In the collaboration of physiologist, pharmacologist, anæsthetist and engineer lies the anæsthetic research of the future.

Embley, could he return, would have no reason for dissatisfaction with the development of his speciality. With the state of the world at large, he would be less content. A junior member of the writer's family, recently invalided from New Guinea, on hearing the title of this address, made the grim comment that, if Embley could return, he would like the present world so little that he would ask the nearest anæsthetist to put him to sleep again. The war in which we are now engaged was not of our seeking and represents merely a hiatus in our scientific progress. The insane nationalism of our age implies, however, a real danger to scientific thought, which should be free, tolerant and international. We live in an age of transition, characterized simultaneously by a violent exacerbation of nationalism and by a social revolution. Whatever world order may emerge from the present struggle, we shall retain our dual obligation; to preserve our freedom of thought as doctors, and our tolerance as members of what was formerly termed the "liberal *bourgeoisie*".

We cannot recall Embley's career without reflecting upon that obscure riddle, the purpose of life. In the words of an old phrase quoted by Carlyle: "Where we are, we know; whither we are going, no man knoweth." The poet Swinburne[30] faced the enigma in the following well-known lines:

> We thank with brief thanksgiving
> Whatever gods may be
> That no life lives for ever,
> That dead men rise up never,
> That even the weariest river
> Winds somewhere safe to sea.

Of the nature of that sea, there is no certain knowing. To Swinburne, it meant oblivion. Faith offers other explanations, but medical men are apt to find faith difficult where no data are. We may feel, however, that no life which adds materially to the sum of human knowledge has been lived in vain. To be remembered with honour after twenty years, to become part of the tradition which moulds the future, is possibly as much immortality as man is justified in expecting. This destiny is Embley's.

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ADDENDUM.

## An Historical Exhibition.

To show the evolution of anæsthesia apparatus up to Embley's day a series of exhibits, drawn in the main from the museum of the Australian Society of Anæsthetists, has been arranged and may be inspected tonight. For comparison and contrast, modern apparatus has been included in the display; a description is given below.

### *1846-1850.*

Ether was given on a folded towel and chloroform on a folded handkerchief. The later change from this "open" method to various forms of inhaler, whilst it reinforced the potency of the anæsthetic, was often a regression from a physiological standpoint.

### *Era of "Cone" Inhalers, 1850-1876.*

The earliest cone was a pinned-up towel containing a marine sponge. Cones were then made with walls of domette (Guy's, *circa* 1870), of leather (Rendle's, *circa* 1870, or the Hyderabad cone of 1890), and finally metal (Südeck's, *circa* 1900). The contained sponge was charged with the anæsthetic, and the cone was applied to the face until a further charge was necessary. As the material of the cone was made less and less pervious, its faults became more and more obvious—restriction of the oxygen supply and accumulation of carbon dioxide. Cone inhalers lingered, especially on the continent of Europe, until the third decade of the twentieth century.

### *Era of "Closed" Inhalers, 1876-1906.*

Chloroform was too potent an anæsthetic to be given by a "closed" method, but the desire for greater anæsthetic potency led to the "closed" administration of ether. The best known of such inhalers was Clover's, which (in its original form, or in modifications by Hewitt, Probyn Williams, Ombrédanne and others) dominated European practice for a generation. The exhibition includes a Clover's inhaler (1876) once belonging to E. H. Embley, and an Ormsby's inhaler (1877). These "closed" inhalers presented ether at its worst, with all the drawbacks of excessive concentration, salivation and mucous secretion, deficiency in oxygen supply and excessive accumulation of carbon dioxide.

### *Era of "Open" Methods of Administration, 1895-1945.*

"Open" ether anæsthesia was reintroduced in 1895, but did not gain general acceptance until about 1905 to 1910. The method was very satisfactory and is still in use, although it is now being challenged by the wide-bored "inhalational" vaporizers of the Oxford type. The wire-framed, gauze-covered masks of Schimmelbusch and Bellamy Gardner, used continuously for the past half-century, are exhibited. So also is a pharyngeal airway; it is curious that so useful and seemingly obvious an appliance was unknown until introduced by Hewitt in 1913. Chloroform, owing to its potency, was always given by the "open" method. The wire-framed, domette-covered masks of Esmarch and Murray, still in use, although perhaps seventy years old, are displayed.

### *Regression to "Semi-Open" Methods, 1905-1941.*

Anæsthetists have attempted, from time to time, to gain anæsthetic potency by concentrating the vapour. To this end, a dome of more or less impervious material was erected around the "open" mask, and such masks have been reintroduced from time to time. The method is really a regression to the old "cone" inhaler, and has the same faults, namely, restriction of oxygen supply and accumulation of carbon dioxide. The display comprises the original semi-closed mask of Ferguson (1905), that of Coutts (*circa* 1930), and an appalling example used by the Italian army in Libya (1941).

### *"Vapour" Methods, 1867-1941.*

Vapour methods, that is, the delivery to the patient of an anæsthetic vapour generated in a mechanical vaporizer, began with Junker's chloroform inhaler (1867). The best known of ether vapour apparatus was Shipway's (1915), used by the British army in the first German war. Both this and Junker's apparatus are represented amongst the exhibits. A Vernon Harcourt's chloroform inhaler (1902) is displayed to indicate the inadequate size of the valves and channels then thought appropriate. For comparison, a modification of Marrett's apparatus (1942) is displayed. It is suitable for trichlorethylene or chloroform, and presents the wide-bored channels and effortless valves which are characteristic of the modern inhalational vaporizer. As the most refined example of this class of apparatus, the Oxford vaporizer (1941) is exhibited; it is fitted, however, with wide-bore valves to replace the unduly small valves supplied commercially. These last were clearly designed for gas anæsthesia by the "semi-closed" method, *circa* 1925, and are inappropriate to an inhalational vaporizer.

### *Endotracheal Anæsthesia, 1871-1945.*

A Trendelenburg's tampon canula (1871) is exhibited as representing one of the earliest attempts at endotracheal anæsthesia. Had it not been forgotten, it might have evolved into endotracheal inhalational anæsthesia as understood today, and as rediscovered by Magill in 1920. When the endotracheal method entered general use in 1909, however, the technique was insufflational, a small-bore catheter and a positive pressure of delivery being used. Boyle's endotracheal apparatus (1912) is shown as typical of this era. Shipway's endotracheal apparatus (1916) is exhibited to illustrate the drip-feed method of generating ether vapour. His gum-elastic catheter and Jackson's laryngoscope are shown, being typical of the period. In 1920, Magill introduced the wide-bore rubber tube, the exhalation valve, the effective pharyngeal pack and the technique of "semi-closed" administration at low pressure. His tubes, elbows, valve and laryngoscope (1930) are displayed in connexion with an appropriate vaporizer (Australian army, 1939). For comparison, a wide-bore endotracheal attachment is shown, of the type used with present-day inhalational vaporizers. A modified form of Cobb's elbow tube (1943) is exhibited to illustrate the technique of intermittent suction drainage of the trachea.

### *Gas Anæsthesia, 1895-1945.*

Modern gas anæsthesia may be said to have begun when effective control could be exercised over the percentage admixture of anæsthetic gas and oxygen. There is no purpose then in showing apparatus antecedent to this period. Hewitt's apparatus (*circa* 1895) is included, however, as being the earliest English gas-oxygen apparatus. The apparatus most used in England to this day is Boyle's, which has been continually improved. An example of the original form (1915), used in the British army in the first German war, is exhibited; it has the pioneer Gwathmey water flowmeters, and once belonged to the eminent Howard Jones, of London. An Australian army gas anæsthesia apparatus (1939) is shown as illustrating the development of the water flowmeter since Gwathmey's day. McKesson's "Nargraf" apparatus (1929) is exhibited as illustrative of the intermittent flow technique with positive pressure; the Austox "D-M" apparatus (1931) is also shown as a local product, employing McKesson's principles. Appliances for carbon dioxide absorption anæsthesia after the method of Waters (1923) are included in the equipment of the army gas apparatus, whilst the McKesson apparatus is fitted with a circle absorber, an appliance first introduced by Sword (1928). The application of the Waters absorber to "controlled respiration" in thoracic surgery (Guedel, 1934; Waters, 1936) is illustrated by appliances in use by Orton in the thoracic surgical unit, Alfred Hospital.

### *Intravenous Anæsthesia.*

When a prolonged intravenous administration is performed merely with syringe and needle, the anæsthetist is immobilized and cannot give due care to his patient. Two devices, conferring mobility and lessening the chances of extravenous injection, are exhibited. One consists merely of the introduction of a flexible rubber tube and of a sight glass or "vein-seeker" between syringe and needle. The other is a syringe holder of the rack-and-pinion type, based upon Rudder's apparatus.

### *Historical Glassware.*

Glassware made and used by Embley in his classic researches at the University of Melbourne is exhibited.

### *Comment.*

The display is intended to trace the evolution of anæsthesia apparatus from early times to Embley's day, most of the exhibits being of his period. Beside them, for comparison

and contrast, are placed certain modern appliances. They are intended to illustrate the progress in recent years in obviating respiratory resistance, in eliminating "dead space" and in assuring an abundant supply of oxygen. Anæsthesia is, however, a living and growing speciality, so that the appliances of 1945 will doubtless appear archaic in 1955. The display indicates, therefore, no finality in design, but merely that conformity with the laws of physiology is essential to sound anæsthetic technique.

*Acknowledgement.*

Being curator of the museum of the Australian Society of Anæsthetists, I am enabled to draw freely upon that collection. For the loan of other apparatus, I am indebted to the Director-General of Medical Services, Australian Military Forces, to Dr. R. Orton and Dr. A. Blaubaum, and to Messrs. Australian Oxygen and Industrial Gases, Limited. For facilities, I am grateful to the Secretary of the Victorian Branch of the British Medical Association and to his staff.

## IMMERSION BLAST INJURIES OF THE ABDOMEN.[1]

By C. KEATING, M.S.M.,
*Surgeon Commander, Royal Navy.*

IMMERSION blast injuries of the abdomen were recognized and described in the last war. My own interest in them dates from the early days of this war, when I was serving at the Royal Marine Infirmary at Deal, and the use of the magnetic mine in the narrow waters of the Channel and the evacuation from Dunkirk provided many cases of these injuries. Later, in the North Atlantic and in the Mediterranean, I had opportunities of observing similar cases, and though stress of circumstances usually prevented post-mortem examinations, I was able to formulate, at least to my own satisfaction, certain guides to diagnosis and treatment.

It should be remembered, of course, that those suffering from immersion blast injuries to the abdomen have been forced to abandon ship, and are likely to have other injuries, such as multiple compound fractures and severe burns. More than once, during the Malta convoys, I have had to deal with patients who had been once rescued, only to be thrown into the water again, and the diagnosis and intelligent observation of such patients (on what has been described as a mobile platform) present certain difficulties.

A good deal has been written concerning the mechanism of these injuries, and attempts have been made to describe in the language of physics and mathematics how the injuries are actually caused. Successful efforts have been made by Wakeley, Williams, Cameron and others to reproduce similar lesions experimentally in animals. This has always seemed a work of supererogation to me, for it is a matter of common experience that when a depth charge or torpedo explodes under water, all those on ships within a distance of two or three miles feel a sensation as if somebody with a heavy sledge hammer had struck a sharp blow on the side of the ship. If one happens to be sitting and resting against the wall of the ship, the feeling is rather as if one were resting in a chair and some malevolent person had kicked the chair smartly in the back.

It is not surprising, therefore, that people immersed in the sea in the actual vicinity of the explosion suffer damage, particularly to the chest and abdomen and their contents, for such people have, in fact, been struck by a water hammer.

If one looks at a series of photographs showing the explosion of a depth charge taken at intervals of three-quarters of a second, one will appreciate the amount of water compression and lateral thrust which must have occurred before such an enormous volume and weight of water could be so violently displaced upwards.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 31, 1945.

### Mechanism of Injury.

The first thing that was observed was the preponderance of abdominal over chest injuries in under-water blast; this was a reversal of the state of affairs which had been found in air blast. It is reasonable to assume that the lungs, enclosed in their relatively rigid bony thorax, should suffer less than the abdominal viscera, protected only by the muscular abdominal wall, when a sudden water-hammer blow is struck. Moreover, most of the patients would be wearing an inflated life jacket, which has been shown experimentally to give a good deal of protection to the chest. Tudor Edwards has suggested that the more rigid chest cages of elderly people give more protection to the enclosed lungs from air blast than the softer and more pliable bony framework of the young.

In underwater blast injuries, in addition to a sudden compression force applied to the chest, there is at the same time a sudden increase in intraabdominal pressure which pushes up the diaphragm. This produces involvement of the pulmonary bases, and the point I should like to make is that the clinical picture arising from the damage to the lung bases is likely, in the early stages, to overshadow the classical signs of the concomitant intra-abdominal damage.

An early balance is, therefore, to be struck between the degrees of intrathoracic damage and intraabdominal damage, and when there is doubt, as there nearly always will be, Cuthbert Wallace's dictum should be applied—that is, "look and see", not "wait and see". Gordon-Taylor has suggested that, because of this dictum alone, Cuthbert Wallace should be canonized and "St. Cuthbert" adopted as the patron saint of abdominal surgeons.

I should like to stress one sign which I have found of use. Although the man with damage to the lung bases holds his abdomen rigid, and limits as far as he can his respiratory excursion, there is a brief moment at the end of expiration when the palpating hand, placed just below the rib margins, can detect a momentary relaxation of the abdominal muscles in those cases in which there is no intraabdominal injury.

I once discussed this sign with Gill, who has encountered a fair number of these early doubtful cases, and he concurred in its presence and extreme diagnostic value in many cases.

All these patients give a history of a sharp pain in the back. Some describe it as being like a kick in the back, others like having a wire tied round their waist and suddenly tightened. This latter sensation is often associated with numbness and tingling of the legs and sometimes with a transient paraplegia, which is particularly unfortunate for the wretched victim immersed in the sea and oil fuel.

Here is a typical history given by a patient whose ship was torpedoed one dark night in 1942:

> After abandoning ship, I swam away to windward to avoid as much oil fuel as possible. I had on a Service lifebelt blown up so that it was easily pressed in. From experience in the *Ark Royal* I had found it was easily punctured if blown up too hard, by bumping into something sharp in the dark.
>
> After about twenty minutes in the water, during which the ship had disappeared, and whilst swimming round collecting the men into groups, there was a sudden explosion, which I judged to be about 100 to 150 yards away. This I thought to be a bomb, although no sound of aircraft was heard. It is hard to describe actually the effect it had on me, in medical terms, but it was just like a band being placed round my waist and being quickly tightened, together with a collapsed feeling, such as the feeling after a sudden diarrhœa evacuation. I decided that if it was a bomb, more would follow, and so attempted to turn over on my back, but found everything had gone numb from the waist down and that I could not raise my legs. This condition lasted for about an hour; then a tingling sensation, like that of pins and needles, set in until the movements of my legs became more or less normal again.
>
> I could not climb aboard the destroyer that picked me up, so was hauled on a rope's end and helped down to the wardroom, but thinking of the lads we left in the sea I went back on deck and did what I could to assist.

> The excitement probably took my thoughts off my own self, for I cannot remember feeling any further effects until after we were landed and I went to bed at an hotel. Then severe griping pains started, particularly when I opened my bowels. These were open at least seven or eight times a day, and it was then I first saw that I was passing bright red blood. This continued for a week, but the slack motions and pain continued for a month before becoming less frequent. The explosion was definitely found to be an enemy's torpedo, which had exploded at the end of its run.

This type of description might be taken as a classical example of the numerous histories given about the immediate effects of underwater explosion in the less severe cases.

Testicular pain is complained of by many patients. Wakeley showed experimentally, using animals (goats, sheep and a monkey), that this is due to small hæmorrhages beneath the *tunica albuginea*. Vomiting is a fairly constant symptom, and is, I think, especially common in those cases in which a perforation or rupture of gut has already occurred. Unfortunately, nearly all of these patients have swallowed oil fuel and sea water, and this complicates the picture.

### Types of Injury.

The type of injury varies from a severe and destructive form causing immediate death, to a mild injury, with no sequelæ other than abdominal pain and shock. All degrees between these two extremes occur, but, as Wolfe has suggested, it is convenient, from the point of view of treatment, to divide patients into three classes.

*Type I.*

The first type covers severe injuries; the patients are moribund on their admission to hospital. They may be suffering too severely from shock to present classical signs of intraabdominal injury, and it can be stated from operative and post-mortem findings that, apart from injuries to the viscera or solid organs, they have all suffered severe retroperitoneal hæmorrhages. Though some of these patients, with the aid of whole blood transfusions, may recover sufficiently to be operated upon, the outlook is very poor, and as such cases rarely occur singly, surgical treatment should be more profitably directed towards patients with the less serious injuries.

*Type II.*

In the second type the amount of visceral damage varies from patient to patient, but in all cases the force has been severe enough to rupture the intestine and occasionally solid organs. These patients are usually suffering severely from shock. The commonest type of injury in a series of 21 cases of my own was laceration of the caecum, of the transverse colon and of the sigmoid at its most dependent part. Two patients presented multiple ruptures. Out of 21 patients operated on, 13 died—a high mortality rate. I think it can be truly said that without operation they would all have died.

*Type III.*

The third type covers the cases in which only a rupture of the mucous and submucous coats has occurred, or there may be an intramural hæmorrhage which subsequently causes pressure necrosis and perforation, sometimes as late as the eighth day. Here is an example.

> A stoker, aged forty years, found himself in the water after his ship had been torpedoed. He was swimming towards a rescue vessel when an underwater explosion occurred, and he felt acute pain in the belly and testicles. He estimated that he was about 100 yards away from the explosion. He found he could not move his legs, and thought he was about to drown when he was picked up. After lying on the deck of the boat for about half an hour, he found he could move his legs again. He vomited four or five times during the next five hours, but the vomitus was not blood-stained. He was given a cigarette, but he found that smoking made him cough, and he was surprised when he coughed up some blood-stained sputum. He reached hospital twenty-six hours after his injury, and was then feeling quite fit. His abdominal pain had completely disappeared, the temperature and pulse were normal. After being in hospital for three days and feeling quite well, he was allowed to go on leave.
>
> During the first three or four days of his leave he felt quite well except for a little abdominal discomfort, but enjoyed his normal life at home. On the eighth day after injury, when he was having breakfast, he had a sudden attack of acute abdominal pain and vomited. When examined two hours later he was sweating and suffering severely from shock. His abdomen was board-like; his condition was diagnosed as a perforated peptic ulcer, and he was removed to hospital. On arrival there his condition had somewhat improved, and the pain became most pronounced in the right iliac fossa, so the condition was thought to be acute appendicitis. Operation was carried out four hours after he reached hospital. The abdomen was then opened through a muscle-cutting incision in the right iliac fossa. Some peritonitis was present, and free purulent fluid was found inside the abdomen. A normal appendix was seen lying over the pelvic brim. The caecum was thickened and rigid, and on its outer wall were two perforations similar in outline and characteristics to the perforations commonly seen in a peptic ulcer. An attempt was made to obliterate the perforations by means of purse-string sutures, but the whole of the caecum was so thickened that this proved impossible. Both perforations were then covered by means of omental grafts. A drainage tube was inserted into the pelvic cavity and the abdominal wall was closed in layers. The patient's condition was never satisfactory after the operation, and he developed bronchitis the following day and died after forty-eight hours. No post-mortem examination was made.

An investigation carried out by Wakeley into eighty cases of underwater blast in which recovery occurred without operation yielded the following results. The commonest symptom was abdominal pain, which persisted from two to three days after the injury to as long as three months in some cases. Melena was present in 82% of these cases and in some persisted for four months. In 20% there was a history of hæmoptysis, and in 14% hæmatemesis was reported. There was no sign of any external injury in any of these cases. Four of these patients had suffered from immersion blast on more than one occasion.

### Treatment.

Blast lesions of the bowel are similar to those produced by bullet wounds; that is to say, the opening tends to close of its own accord owing to the prolapse of the mucous membrane. In some blast injuries of the gut a laceration of the bowel wall and a split in the mesentery are present. In nearly every case the omentum makes some attempt to cover and seal the laceration, and it is quite likely that some patients have recovered without operation, because of the protective mechanism of the great omentum. However, if there is any doubt whatever in a case of blast injury of the abdomen, it is far safer to perform a laparotomy and to "look and see", rather than to "wait and see"; for if there is delay the patient may die of peritonitis.

The treatment of the actual perforations may necessitate excision in the most severe cases in which extensive laceration of the bowel wall has occurred, but in the greater number of cases, in which laceration is not extensive and in which there is considerable prolapse of the mucous membrane, a more conservative suture operation may be undertaken with success. The prolapsed mucous membrane is united. A few Lembert sutures are put in over the area to reinforce the suture line and prevent leakage or adhesions to that area of the bowel. If excision of the bowel is necessary, as it may be when the wall is completely torn across and the mesentery injured, then after the torn edges of the bowel have been excised the two ends may be sutured so that end-to-end union is obtained. This, however, is not always possible, owing to difference in size of the two ends of the bowel. In such cases the ends of the bowel have been invaginated and sutured, a lateral anastomosis performed and the line of the anastomosis reinforced by an omental graft.

It is usually advisable to give the patient a blood transfusion during the operation, if there has been much loss of blood. In every case in my experience extensive retroperitoneal hæmorrhage has occurred and the blood volume has been reduced, but there has been no hæmoconcentration.

In some of the cases dealt with there have also been extensive burns of the hands and face from burning oil, as well as intraabdominal damage. In such cases it has been necessary to give a plasma transfusion first, because of the hæmoconcentration, and to follow this with a whole blood transfusion. Blast injuries of the abdomen in association with compound fractures of the long bones are not uncommon. It is hardly necessary to add that such cases need a very critical clinical eye, if a successful issue is to result.

In those cases in which the caecum was ruptured—and these were common, possibly because the caecum frequently contains more gas than any other part of the large bowel—the laceration was invariably situated on the outer side of the caecum. It is generally known that rupture of the caecum may be a complication of annular carcinoma of the sigmoid colon, and in such cases the rupture takes place at the outer border of the viscus, just lateral to the longitudinal band. It is curious how closely these lesions, produced by two different kinds of pressure, simulate one another.

The laceration of the caecum due to blast is not, as a rule, extensive, and the mucous membrane is prolapsed in an attempt to close the rent and prevent extravasation of the caecal contents. In most cases the omentum plays its part in an attempt to seal the laceration, and a localized abscess develops. It seems to be rare for general peritonitis to develop in these cases, unless an extensive rent has been produced. As the damage is always on the outer side of the caecum, the same tendency to shut off the inflammation occurs as in cases of appendicitis.

> I have seen one patient a week after he was rescued from the sea after being in the vicinity of the explosion of a mine; he was suffering from pneumonia following the blast injuries to his lungs, and had an abscess of the outer side of the caecum. He was treated conservatively, and after the pneumonia had been cured the abscess was opened and drained. Whilst this was being done, the omentum, which was found to be enveloping the caecum, was detached and a healed rent in the outer border of the caecum was discovered. The patient made an excellent recovery from all these blast injuries.

Post-operative treatment has been on routine surgical lines for any injury to the gut, except perhaps that rather large doses of morphine have been given. The after-history of these patients seems to be usually uneventful; but one patient who had been operated upon in December, 1939, for a blast injury of the sigmoid colon, and had made a good recovery, subsequently in October, 1940, developed acute intestinal obstruction and was successfully operated upon a second time. The cause of the obstruction was then found to be a band passing between the terminal portion of the ileum and the sigmoid colon; a loop of small intestine had been nipped under this band. It seems likely that other similar cases will arise in the future.

### Prevention.

Swimming on the back is popularly believed to reduce intraabdominal damage, and experimental work with animals confirms this to some extent. An inflated life-belt worn low also gives complete protection, but it considerably alters the centre of buoyancy. Probably a rubber sponge abdominal and loin pad would give the best results; but there would be from the sailor's point of view disadvantages in wearing such appliances.

## BLAST INJURY OF THE BRAIN.[1]

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THE effects of blast are strange, and during the heavy "blitzing" of Britain were often commented upon. Some time after an air raid on Plymouth a pet tortoise was missing from a garden in a bombed area. It was later found on a roof 25 feet above the ground, a piece of its carapace missing, but otherwise apparently none the worse, and it is now back in the garden. It is well known that windows quite near a bomb-burst are sometimes spared from damage while others farther from the explosion are shattered.

The central nervous system of those in the vicinity of an explosion may be affected, and structural changes may be produced in the brain or cord. Probably this has occurred more often than is realized.

### The Effect of Blast on the Brain.

The following case illustrates the effect of blast on the brain.

CASE I.—In July, 1943, a seaman, aged nineteen years, was standing by a four-inch gun. Upon its discharge he noticed a sudden stiffness and numbness in the left hand and arm, which lasted for about ten minutes and then recovered completely. Three weeks later began the first of a series of attacks of numbness and tingling, which, starting in the left hand, spread to the elbow, shoulder and side of the face, and were accompanied by involuntary abduction and elevation of the arm, spasm of the face and loss of consciousness for a few minutes. On recovery there was some residual weakness of the left upper extremity, which gradually increased and spread to the leg, and seizures became more frequent, occurring four to six times per day. The patient was discharged to hospital suffering from a left spastic hemiparesis and hypæsthesia to pin prick and light touch over the left side, and the observation was made that the condition was possibly hysterical. He stated: "All my trouble started when the big guns fired." He was investigated in a neurosurgical unit in the north, and air studies were made. As the position, size and shape of the ventricles were regarded as normal, he was discharged from the unit. He was then admitted to the Naval Neurosurgical Unit at the Royal Naval Auxiliary Hospital, Sherborne, and reinvestigated, the air studies being repeated. These showed an apparently normal ventricular system. The visual fields were full and the cerebro-spinal fluid was normal. The electroencephalogram revealed asymmetry in the cortical rhythm from the two hemispheres, a slower frequency being present in the left temporo-occipital region. An outstanding feature in the case was the fact that the left plantar reflex was certainly extensor in type. It was decided that there must be a cerebral lesion to account for the pyramidal signs, and after consultation with Surgeon Lieutenant-Commander E. Buzzard, Royal Naval Volunteer Reserve, under whose care the patient had been admitted to the unit, it was decided, despite the normal ventriculographic findings, to explore the post-central area, and a bone flap was accordingly turned down in this region. The dura in the posterior part of the field was bulging, and when it was opened the cerebral gyri here were green in colour, widened and elevated over an area about the size of a penny. On incision of the brain in this area a shallow but extensive cavity was entered containing old blood clot. This was removed, and the patient made an uninterrupted recovery; some residual left-sided weakness and asterognosis, however, were still present at the time of his discharge from hospital. It is interesting to note that when the ear drums were examined by an otologist he reported: "The condition is compatible with a mild trauma to the ears from the time of the gun blast."

*Comment.*

There can be little doubt that this intracerebral hæmorrhage was consequent upon the gunfire. Somewhat similar cases were reported by Mott (1919) in the last war, the lesions being discovered *post mortem*. Similar post-mortem findings have also been described in this war (Tunbridge and Wilson, 1943; Ashcroft, 1943), but I cannot trace a previous case to the one here reported in which the lesion was exposed at operation.

Experimental work by A. Mairet and E. Durante (1917) on rabbits showed that hæmorrhages were produced on the surface of the cord and in the grey matter of the cortex and bulb when the animals were subjected to the near effects of high explosives.

### Apathy.

In 1941, Stewart, Russell and Cone described the curious behaviour of a pheasant found after an air raid. The bird

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 31, 1945.

was in a dazed condition resembling catatonia. There were no external injuries, but hæmorrhages were present in the lungs, heart muscle and brain. Capillary congestion was present through the cerebrum, and gross hæmorrhages had occurred in the forebrain. When found, twelve hours after an air raid, the pheasant was ninety feet from the edge of a bomb crater and was standing on both legs with its eyes closed. It was picked up in this attitude, and when pushed forwards would take only a few unsteady steps.

It may be remembered that Flourens's pigeons, from which he had removed the cerebral hemispheres, were able to fly and to superficial examination appeared normal birds, but that they were unable to initiate movements and would stand indefinitely in a dazed way. In this respect these birds, from which Flourens drew his fallacious conclusion that the brain acted as a whole and that localization of function did not occur in it, may be compared with the pheasant with damaged cortical connexions from the blast-produced hæmorrhages.

In common with others, I have noted the apathy, lassitude and dejection of certain of the human victims of air raids.

CASE II.—In an air raid on Bristol a bus conductress was blown off the platform of her bus which had a direct hit by a bomb, all the passengers being killed. I saw her in hospital soon afterwards. There were no external injuries, but she was apathetic and could not be persuaded to speak, although she was quite conscious. The only response she made to examination was when she was told that the aircraft which had bombed her bus had been shot down. Her eyes became more lifelike, and she remarked: "A damn good job, too"; but then again she lapsed into her condition of apathy which persisted for some days.

*Comment.*

Lumbar puncture in some of these cases has yielded blood or xanthochromic fluid. Small scattered hæmorrhages in the central nervous system may have occurred as a result of blast more frequently than has generally been recognized, and may have been responsible for the apathy of these patients. Apathy has been a striking feature of many air-raid victims, and it will be recalled that it was shown by Stewart, Russell and Cone's pheasant.

### The Mechanism of the Cerebral Lesions.

It has been suggested that the mechanism responsible for the production of the cerebral lesions is the hydraulic-like pressure transmitted to the cerebro-spinal axis in its closed bony walls by sudden compression of the thoracic cage. Such compression must produce violent back pressure on the venous side of the circulation. A sudden rupture of vessel walls may conceivably be caused by the decompression which suddenly succeeds the compression wave. It will be noticed, however, that in Case I an interval of three weeks intervened between the initial symptoms produced by the gun blast and the seizures and hemiparesis, so that this was probably an example of "*Spätapoplexie*". The pathogenesis of delayed traumatic intracerebral hæmorrhage has long given rise to speculation. It has been suggested that hæmorrhage may result from foci of softening produced as a direct result of injury, that it may arise from preexisting alterations in the vascular bed, or that it may occur as a late leakage from a lesion from which primary hæmorrhage occurred at the time of injury. Vessel damage with diapedesis and escape of blood into the tissue of the brain may well be the initial lesion produced by the blast wave, and progressive changes following such damage may subsequently lead to a break in the vessel wall and a delayed and more massive intracerebral hæmorrhage. Bollinger (1891), Marburg (1928) and others have suggested such a mechanism as the cause of "*Spätapoplexie*" from head injury caused by the more usual forms of violence, and it may conceivably be the explanation of the delayed symptoms produced by blast as in Case I.

### Summary and Conclusions.

Blast may give rise to hæmorrhages within the substance of the brain. These have been demonstrated at operation and at autopsy and have been produced in laboratory animals. They may be multiple, small and scattered, or more extensive and localized, when they may give rise to focal signs. They are probably produced indirectly as a consequence of compression of the chest by the blast wave, and may occur immediately or be delayed in onset for as long as several weeks.

### Acknowledgement.

I am indebted to the Medical Director-General of the Navy and the Surgeon Rear Admiral in charge of a naval hospital for facilities for operating on one of the patients referred to.

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## INJURIES TO THE EAR DUE TO BLAST.[1]

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BLAST INJURIES to the ear cause deafness which falls into three clinical types: (i) middle ear deafness with or without rupture of the ear drum, (ii) an abrupt high-tone loss due to blast concussion, and (iii) a gradual high-tone loss due to slight concussions in series over a long period. All three are accompanied by tinnitus.

If there is a rupture of the drum it is usually in the anterior aspect, and the margins of the perforation are frequently everted. This is said to be due to the reactionary suction component of the blast wave. There is always slight hæmorrhage, which may not be sufficient to reach the external meatal opening and so may not be noticed until a proper examination is made. There is also temporary deafness of the middle-ear type, which will recover if no infection is present. This recovery period has been estimated by various observers as from fourteen to fifty-six days.

There is one type of case in which complete recovery never occurs—that is, the case in which there is a tendency towards progressive conduction deafness, or otosclerosis. It is probable that such patients have a mild preexisting deafness which they have not noticed. In my own experience I have found that they date their deafness from the explosion, and that subsequently the progression is rapid.

In cases in which no rupture of the drum has occurred, a varying degree of hyperæmia is evident, beginning along the handle of the malleus and extending over the rest of the drum as it grows more severe. Occasionally it can be seen for nearly a quarter of an inch along the meatal wall. This hyperæmia may be so intense that the vessels burst and a small hæmorrhage takes place inside or outside the drum.

In these milder cases the middle ear deafness recovers rapidly; but cochlear damage is more likely to be present as well, when the drum does not rupture. This is shown by a high-tone loss, which will be discussed at a later stage. The tinnitus often lasts longer than the deafness and may even be permanent in some degree.

From the foregoing it will be seen that the main principle to be observed in the treatment of trauma of the ear drum is prevention of infection. Syringing out of the ear, in spite of the strictest instructions to the contrary, is still

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 31, 1945.

being carried out, usually by medical orderlies who are not under proper supervision at the time. In nearly every case in which this is done, infection supervenes. It is a recognized treatment, as will be realized from later quotations in this paper, to insufflate sulphonamide powder into the meatus after rupture of the drum. I have encountered a number of cases in which this has been done, and my impression is that a piece of sterile cotton wool packed firmly into the meatus is still the best treatment, and that the blowing in of the powder may push in infected material as well. You will all have your own ideas on this point.

It is most important to instruct patients not to blow air through their Eustachian tubes while the rupture is unhealed and uninfected. Occasionally one sees a wad of wax in a meatus which has been bleeding after blast injury, and cases have been observed in which the wax has been tunnelled through and portion of the drum is visible. If the wax cannot be removed by hook, spoon or forceps, it should be left for about two or three weeks, unless, of course, there is evidence of infection behind it.

Palmer quotes sixty cases of blast perforation in Indian troops following a land-mine explosion, and makes the following points: (i) the antero-inferior quadrant is the commonest site of injury; (ii) the proportion of infections, under the most ideal conditions, is 35% to 40%; (iii) insufflation of sulphonamide powder is probably of value in preventing infection; all the patients were treated by this means; (iv) deafness is of the middle-ear type, superimposed on a temporary nerve deafness; (v) the subsequent damage to hearing is not severe. These patients were examined soon after being injured.

Tunbridge and Wilson, reviewing the literature on blast injuries, found that in all the cases in which the ear drums were examined soon after exposure to blast, a varying degree of congestion was present. In very mild cases a slight dilatation of the vessels around the handle of the malleus is present, which subsides rapidly. As the severity of the injury increases, so does the degree of congestion, going on to hæmorrhage and rupture of one or both drums. The degree of deafness also depends on the severity of the injury, and the more severe the deafness, the longer it takes to recover. Tunbridge and Wilson quote ten to fifteen days as the recovery period, and they do not differentiate the types of deafness, though they state that some of their patients had permanent loss of hearing to some degree. These same observers state that in one series of cases (the number is not quoted) all the patients with ruptured drums were treated with sulphonamide powder, and in no case was there evidence of infection.

Silcox and Schenck, in a report on the casualties aboard a hospital ship, state that the causes of the damage are (i) atmospheric blast from explosions, (ii) immersion blast from explosion of depth charges when the patient was in the water. The severity of the injury depends on the following factors: (*a*) the proximity of the source of sound, or the centre of the detonation; blast waves in water are about four times as effective as in air; (*b*) the duration of exposure to the stimulus; (*c*) the presence of previous aural disease; this increases the liability to injury in all cases; (*d*) the use of protective devices. If the drum is ruptured, the degree of permanent deafness tends to be diminished, as it lessens the impact on the inner ear.

In abrupt high-tone loss, a sudden, sharp drop occurs in the audiogram between 1,000 and 2,000 cycles, or between 2,000 and 4,000 cycles. As a rule, the more severe injuries produce a loss in the lower frequencies, and though the audiogram of these patients may reveal a slight rise towards the top of the scale, the hearing, from the sharp initial drop to the upper limit, is much diminished. Both ears are usually affected on the same pattern with minor variations, but cases have been observed in which one ear alone has been damaged. Tinnitus is frequent, and occurs characteristically at nearly the same frequency as that at which the abrupt fall in the audiogram is situated. In a small proportion of these cases a temporary labyrinthine irritation is present.

Gradual high-tone loss comes from exposure to any constant or intermittent noise; for example, gunfire over a period of years produces such a condition in many cases. Wilson has published two papers, in which he gives the results obtained with 85 and 108 recruits in two different experiments; he gave them examinations and plotted their audiograms before and after a course of musketry. His results indicate that persons who already have some impairment of hearing or have had aural disease, and persons whose ears are easily fatigued, are more susceptible to acoustic trauma than normal persons.

The fatigue test is made by exposing each ear in turn to a 2,048 cycle tone of an 80 decibel intensity for eight minutes. The audiogram reading is taken just before the fatiguing note and two minutes after, only the 4,096 threshold being used as a guide, since this frequency perception is found to be easily fatigued by the 2,048 tone. These observations represent a means of determining whether applicants for work in noisy industries are likely to have their hearing affected detrimentally.

Most of the audiograms of those patients with a gradual high-tone loss reveal a "dip" around the 4,000 cycle mark, with normal hearing higher up the scale; but in severe cases there may be a gradual depression from the low tones right up to the highest pitch.

The consensus of opinion of a number of observers suggests that the deafness due to abrupt high-tone loss remains stationary, and that gradual high-tone deafness tends to be progressive, even when the inciting trauma is withdrawn.

The pathology of these two types of perception deafness is as follows, according to Negus. In the abrupt type, the lesion is hæmorrhage into the labyrinth. In the gradual type, a general degeneration of the organ of Corti occurs, and is greatest in the basal coil of the cochlea at a point corresponding to the 4,000 cycle frequency.

### The Use of Protective Devices.

The protective method in general use in the Royal Navy is the insertion of dry cotton-wool plugs into the meatus, and observers state that there is no evidence that these provide any protection. Dickson and Ewing, discussing their investigations into the protection of air crews against aeroplane noise, make the following statement:

> No device is effective unless it is completely tight fitting. Sound penetrates through minute apertures, hence the very small amount of protection given by plugs of dry cotton wool, which is somewhat harsh, as well as porous, and therefore, for both reasons, leaky.

Somewhat greater protection is given by well-greased cotton wool and by various forms of rubber plugs. However well plugged the external auditory meatus may be, the protection is limited; moreover, effective plugging lessens the ability to hear orders. If meatal plugs give only moderate protection against prolonged exposure to noise, the protection they give against a blast wave must be very little. Protection against aeroplane noise has been made more effective by the use of the high-altitude flying helmet, in which earphones form part of the protection. It is probable that protection of gun crews can be improved by the development of an anti-flash helmet containing earphones, which will give some protection to both the ear drum and the side of the skull.

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# OBSERVATIONS UPON THE "CRUSH INJURY" SYNDROME AND VOLKMANN'S CONTRACTURE ASSOCIATED WITH SEVERE BRAIN INJURY AND HYPERTHERMIA.

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IN an investigation of the victims of London air raids, Bywaters, Delory, Rimington and Smiles (1941) found that the red or brown pigment excreted in the urine in the "crush injury" syndrome was not hæmoglobin, but the muscle pigment, myohæmoglobin. Minami (1923), on the basis of German observations during the 1914-1918 war, and later Gilmour (1941) and Bywaters and Beall (1941), had previously drawn attention to the similarity of the crush syndrome to equine paralytic myohæmoglobinuria.

Bywaters and other authors have pointed out that a syndrome similar to that following crush injury can develop after motor car accidents with damage to the blood supply of muscle but without prolonged crushing, if isolated muscle fibre necrosis is caused (for example, by arterial spasms). Several such cases have been recorded in the literature (Husfeldt and Bjering, 1937; Bywaters, Delory, Rimington and Smiles, 1941; Bywaters and Beall, 1941; Cohen, 1941; Lodge, 1941; Bywaters, Graham and Clarke, at a discussion at the Royal Society of Medicine, 1942; Bywaters and Dible, 1942). Bywaters, during the discussion at the Royal Society of Medicine in 1942, stressed the importance of spectroscopic investigation of the urine for differentiation between myohæmoglobin and hæmoglobin:

> These cases . . . closely resemble cases of crush injury clinically, biochemically and histologically . . . but it is still uncertain whether the pigment excreted in the urine is hæmoglobin or myohæmoglobin; it is obviously a very important point. Such cases must occur more frequently . . .

In the same discussion Belsey drew attention to Volkmann's contracture as one of the conditions in which the "crush syndrome" might be expected to occur, but in which no renal impairment had yet been described, conceivably because minor degrees of renal damage had failed to attract attention.

A case is now to be described in which the observation of myohæmoglobin in the urine enabled a relatively mild "crush syndrome" to be detected in a child who had been injured by a motor car and had suffered a compound fracture of the skull, a crushing injury of the right forearm followed by Volkmann's contracture, and lacerations with much muscle damage to the lower part of the right thigh.

## Report of a Case.

The patient, a boy, aged nine years, was admitted to the Royal North Shore Hospital of Sydney at about 8 p.m. on October 7, 1944, having been knocked down by a motor car. He was unconscious and lying in an attitude suggesting cerebral irritation. His breathing was stertorous. The colour of his skin was pale, but the lips and extremities were cyanosed. The lids of the right eye were bruised and œdematous. A lacerated wound, about two inches long, was present in the right fronto-parietal region of the scalp, in the depths of which a comminuted fracture of the skull could be felt. There was a blood-stained discharge containing cerebro-spinal fluid from his nose and both ears. The right upper limb had severe contusions, and a lacerated wound with a surrounding area of contusion was observed in the upper part of the right popliteal fossa. Minor contusions were present in the right leg and left thigh. The blood loss prior to his admission to hospital could not be ascertained; but although none of the wounds was bleeding when he was examined in hospital, a condition of primary shock was present, the systolic blood pressure being only 80 millimetres of mercury. The pulse rate was 130 beats per minute, the respirations numbered 32 per minute, and the temperature was 100·6° F. The pupils were equal and small, and did not react to light. The knee jerks and other reflexes were absent.

The lacerated wounds were cleaned, dressed with sulphanilamide powder and sutured. A prophylactic dose of tetanus antiserum and gas gangrene antiserum was given intramuscularly. Four ounces of a 50% solution of magnesium sulphate were instilled into the rectum and an intravenous infusion of a 10% solution of glucose in normal saline solution was commenced. Sedation was brought about by an intramuscular injection of "Dial", and sulphonamides were given by the same route for prophylactic purposes. An hourly record of the temperature, pulse rate and respiration rate was commenced (Figure I) and continued.

During the next sixteen hours little change occurred in the patient's condition. He was incontinent of urine and took no fluids by the mouth. The condition of the temperature, pulse and respiration can be seen from Figure I. Röntgenological examination of the skull revealed "a slightly depressed fracture of the right fronto-parietal region and a fracture of the base extending across to the middle fossa on the left side".

On October 9 the patient was still unconscious, although moving all four limbs. Only slight neck rigidity was noted. The plantar reflexes were now extensor in type. From both ears blood-stained fluid was still being discharged. Spinal puncture was performed and the cerebro-spinal fluid was found to be under a pressure of 225 millimetres. There were 3,000 red cells in each cubic millimetre; the protein content was 200 milligrammes *per centum* and the chloride content was 480 milligrammes *per centum*.

Later, the temperature rose to 107° F., the breathing became more laboured and the pulse rate was uncountable. As the blood count revealed only 7·5 grammes of hæmoglobin per 100 cubic centimetres of blood and only 3,000,000 red cells per cubic millimetre, the intravenous infusion of saline solution with glucose was replaced by blood transfusion, 500 cubic centimetres being given. The hyperthermia was treated by the administration of ten grains of aspirin dissolved in water into the patient's stomach through a Ryle's tube and by the instillation of six ounces of iced water into his rectum; these measures were followed at intervals by spongings with iced water until the temperature had fallen to 104° F.

On the following day the patient's condition had improved, although he was still unconscious and the plantar reflexes were still extensor in type. Urine was passed into the bed at intervals, but it was quite clear.

On October 11 further improvement had occurred and consciousness was regained; but the urine passed into the bed was noticed to be dark brown in colour. A small amount was obtained for spectroscopic examination, which showed that it contained a large amount of "methæmoglobin" and some urobilin. After this, retention of urine occurred and catheterization at intervals of six hours was necessary. The patient was able to take fluids by the mouth, so that sodium bicarbonate solution was given at intervals of two hours until the urine became alkaline, when the intervals were increased, yet kept short enough to maintain the alkalinity of the urine.

The next day the patient's condition had deteriorated again. The temperature had risen to the vicinity of 102° F. and the pulse rate to 140 beats per minute. The systolic blood pressure had fallen to 100 millimetres of mercury and the patient was in a condition of secondary shock and unconsciousness, from which he could not be roused. Some blood-stained discharge was still coming from the ears. The blood urea level was found to be 57 milligrammes per 100 cubic centimetres and the carbon dioxide combining power of the plasma 42 volumes *per centum*, while the plasma protein content was practically normal (5·4 grammes per 100 cubic centimetres). The urine, which was being excreted in small amounts, had a deep brown colour and was alkaline. It contained large amounts of oxymyohæmoglobin (53 milligrammes per 100 cubic centimetres). The condition was now characteristic of the syndrome of "crush injury". In view of the relapse into unconsciousness, lumbar puncture was repeated, but the pressure of the cerebro-spinal fluid was only 50 millimetres, which is very low. The fluid was only faintly blood-stained and contained 8,000 red cells per cubic millimetre. The head of the bed was lowered and an intravenous infusion of a 10% solution of glucose in normal saline solution was started. Within two hours consciousness was regained.

On October 13 further improvement had occurred in the patient's condition. He was fully conscious and able to take fluids by mouth. The urine was a much lighter brown in colour; myohæmoglobin was still present, but the concentration had fallen to 18 milligrammes *per centum* and urobilin was now present in considerable quantities. The discharge from his ears had practically ceased and the plantar reflexes were no longer definitely extensor in type. For the first time, however, a Volkmann's contracture of the right forearm was noted. This prevented full extension of the fingers and elbow. The opinion of Dr. A. R. Hamilton, honorary orthopædic surgeon to the hospital, was sought, and on his advice individual straight metal splints were applied to each finger and a long cock-up splint was applied to the wrist. Massage and passive stretching of the affected muscles and digits were also instituted.

The contusions to the muscles of the right thigh had subsided, but the lacerated wound behind the right knee, although clean, was gaping widely and filling with granulation tissue.

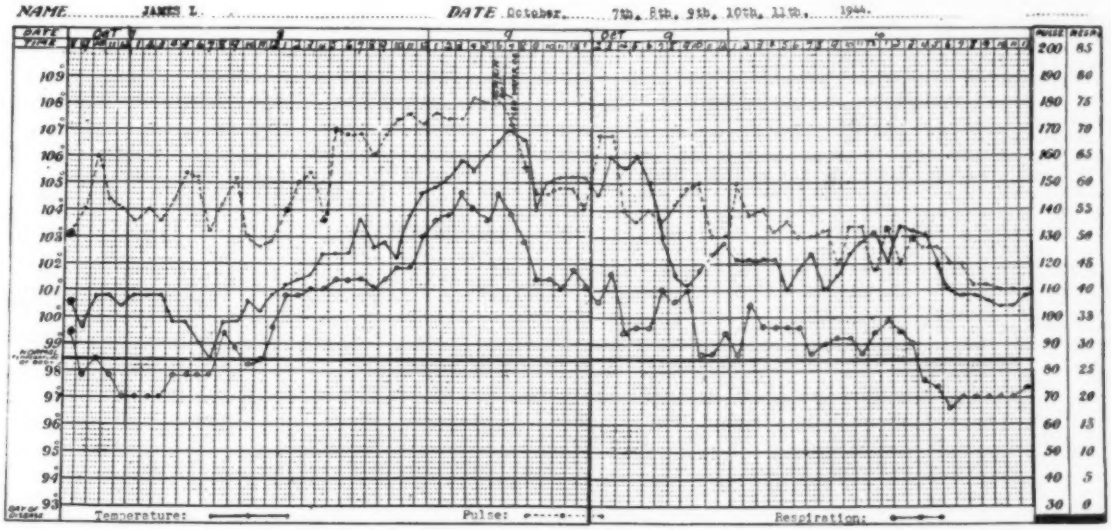


FIGURE I.

From October 14 onwards progressive improvement occurred in the patient's condition. He became continent of urine and it was then possible to measure the amounts which were being passed in increasing quantities. The myohæmoglobin content was only 12 milligrammes *per centum*, and by October 18 no myohæmoglobin was present, although urobilin was still detected in considerable quantities. By October 23 neither myohæmoglobin nor urobilin was detectable in the urine.

On October 31 a split-skin razor graft was applied to the unhealed portion of the lacerated wound behind the right knee, from the posterior surface of the right thigh higher up. The contracture in the flexor muscles of the right forearm responded well to treatment; the skin graft took completely, and the patient was discharged from hospital, symptom-free, on November 22, 1944.

In the absence of any gross depression in the fracture of his skull no further operative interference to his head is contemplated.

### Isolation of an Abnormal Urinary Pigment.

An abnormal urinary pigment was isolated as follows.

Five hundred cubic centimetres of urine were slightly acidified with acetic acid and repeatedly extracted with chloroform. The combined extracts were concentrated to a small volume by distillation, and the chloroform solution was extracted with dilute ammonia. The solution was red and showed an absorption band at 542 millimicrons. Zinc acetate did not produce the fluorescence characteristic of zinc-urobilin. Acid changed the colour to yellow.

After acidification with acetic acid the yellow pigment was extracted with ether; a 10% concentration of hydrochloric acid extracted little from ether, a 20% concentration of hydrochloric acid extracted somewhat more. The reddish solution in hydrochloric acid showed an indistinct absorption band in the blue-green. From the acid solution the pigment could be taken up again by chloroform and had a reddish colour, which by washing with water was changed to yellow. Sodium hydroxide extraction from ether produced a red colour; the absorption band was at 548 millimicrons.

No similar pigment could be obtained from the urine of other patients which contained urobilin. Little urobilin was extracted by chloroform from the nearly neutral solutions; it required the addition of hydrochloric acid to make the urobilin extractable by chloroform. From the urine of one patient with liver cirrhosis a small amount of pigment of the same behaviour and spectroscopic properties, in the form of clusters of thin yellow rectangular leaflets, was obtained by evaporation of the ether solution.

### Discussion.

The late onset (fourth and fifth days) with oliguria and anuria, the passing of acid urine containing myohæmoglobin, lowered alkali reserve of the plasma and increased blood urea content are characteristic features of the syndrome. The initial shock may have been connected with the head injuries, but the secondary shock on the fifth day is an unusual feature. In this case the crushing of the muscles of the right thigh and of the right forearm contributed to the syndrome. The mild degree of uræmia and acidosis indicates that the renal impairment was not great. By appropriate therapy the urine was rapidly rendered alkaline, and gradual improvement in the urinary excretion occurred.

The recognition of myohæmoglobin was based on the position of the α band of oxymyohæmoglobin in the Hartridge reversion spectroscope. This was found at 582 millimicrons, while that of human oxyhæmoglobin was at 578 millimicrons, in agreement with the findings of Bywaters. The first specimen of urine (collected on the fourth day, during the period of incontinence) was brown and strongly acid (pH 5·7). It contained not oxymyohæmoglobin, but the ferric compound, together with an "acid hæmatin" precipitate. Hence the presence of methæmoglobin was reported. The following morning a fresh catheter specimen of urine contained oxymyohæmoglobin, and a reinvestigation of the first specimen, which had been kept, showed that the soluble brown compound in it had been metamyohæmoglobin. After reduction with a minimum of sodium hyposulphite followed by oxygenation the absorption band was that of oxymyohæmoglobin (582 millimicrons).

The first sample of urine was still acid after the addition of one cubic centimetre of 1% sodium carbonate solution

to five cubic centimetres of urine. Dilute sodium hydroxide solution precipitated extraordinarily large amounts of typical crystals of triple phosphate. The precipitate contained a little calcium, but consisted mainly of magnesium ammonium phosphate. This observation appears to be of importance. Duncan (unpublished results quoted by Bialock and Duncan, 1942) also noted tremendous elevations of inorganic phosphorus in dogs with experimental crush injury. Dunn, Gillespie and Niven (1941) have drawn attention to the similarity of the renal damage in "crush injury" to that produced experimentally by feeding phosphate to rats (McFarlane, 1941), the lesions being restricted to the ascending limbs of Henle's loops and the second convoluted tubules. Tissue autolysis may set free considerable amounts of phosphate and lactate. The phosphate content of the blood was found to be increased in "crush injury" (Bywaters, 1942) as well as in equine paralytic myohæmoglobinuria (Grzycki, 1934, quoted by Bywaters, Delory, Rimington and Smiles, 1941). It thus seems that more attention should be paid to the phosphate content of the urine in future studies of "crush injury".

The pathogenesis of the renal impairment in "crush injury" is not yet understood. Weighty evidence has been brought forward against the hypothesis that the products of acid denaturation of myohæmoglobin cause the damage by mechanical blockage of the tubules. Were this hypothesis correct, the urine passing through the unblocked tubules would be expected to be normal; but in fact it is a poorly concentrated glomerular filtrate. Myohæmoglobinuria was at first considered to be a symptom of muscular destruction rather than the cause of the renal damage. Recent experiments, however, indicate a closer connexion between myohæmoglobin excretion and renal damage. Bywaters and Popják (1942) found that by experimental crush injury some symptoms similar to those of "crush syndrome" could be produced in the rabbit, but that in this animal (whose muscles do not contain myohæmoglobin) no serious organic lesions could be observed in the kidney. (It may or may not be accidental that there was also no increase of phosphate in the blood of the rabbit.) On the other hand, intravenous injections of human myohæmoglobin into the rabbit produced renal damage (Bywaters and Stead, unpublished results quoted by Bywaters, 1943). There is an undeniable similarity between the renal damage in "crush injury" and that caused by incompatible blood transfusions; in the latter also the renal impairment does not appear to be caused by mechanical blockage. Eggleton, Richardson, Schild and Winton (1942, 1943), experimenting with dogs, found evidence against the hypothesis that a toxic substance was released from the injured limb, transmitted through the blood stream to the kidney, passed through the glomerulus and concentrated in the tubules, there to exert its effect. They came to the conclusion, however, that no single factor could explain the damage.

In our case the amount of urobilin excreted in the urine was increased, and the excretion of urobilin persisted even longer than the excretion of myohæmoglobin. The Schlesinger test (green fluorescence of the complex zinc compound) was applied, traces of alcoholic iodine solution being added to oxidize urobilinogen to urobilin. It is doubtful whether the urobilin found in the urine of the patient was a breakdown product of myohæmoglobin. Myohæmoglobin has a clearance twenty-five times as great as that of oxyhæmoglobin (Yuile and Clark, 1941). In typical "crush injuries" neither myohæmoglobin nor bilirubin occurs in the plasma in demonstrable amounts. It is even uncertain whether in man myohæmoglobin is catabolized to bilirubin and urobilin. Whipple and Robscheit-Robbins (1926) reported an increase in bile pigment formation after the intravenous injection of myohæmoglobin into dogs, and Carlström (1939) in equine paralytic myohæmoglobinuria; but in progressive muscular dystrophy and during reinvolution of the uterus after childbirth, Meldolesi, Siedel and Möller (1939) found no excretion of urobilin in the urine, but a transformation of myohæmoglobin into myobilin, the polypeptide compound of a yellow dipyrrolic substance, which was excreted in the fæces. It appears more likely that the urobilin in our case was derived from hæmoglobin. Hæmatomata were present in the right thigh and forearm, and the occurrence of internal hæmorrhages elsewhere was also made likely by the low figures for hæmoglobin and red cells, which do not appear to be adequately explained by external hæmorrhage during the accident, or by the saline infusions.

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## Reports of Cases.

### A CASE OF Q FEVER IN NORTH QUEENSLAND.


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AND

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BETWEEN September, 1935, and August, 1944, 217 cases of Q fever were diagnosed in the laboratory of the Queensland Department of Health and Home Affairs. All these patients, with one possible exception, lived in south-east Queensland, between Gladstone (latitude 24° south, longitude 151° east)

and the New South Wales border. The possible exception had worked both in Brisbane and in Cairns, North Queensland.

The 218th patient was a resident of Ingham, North Queensland (latitude 18° 40′ south, longitude 146° east).

### Clinical Record.

J.O., aged thirty-nine years, was admitted to Ingham District Hospital on August 28, 1944. His occupation was that of turncock for the water authority. He moved about in drains and in undergrowth near the river bank. He had no recollection of bites from ticks or other vermin, nor did he show any sign of bites. He had not been out of the Ingham district for years. He had had no previous illness.

Four days before his admission to hospital he became ill with fever, and cough with blood-stained sputum. The clinical picture was typically that of lobar pneumonia and treatment was begun with sulphapyridine. There appeared to be an immediate response, the temperature falling to normal the next morning. However, it rose again and remained high for a week, with large remissions and heavy daily sweats (Figure I). The pulse rate was comparatively slow during this time. He had severe headache throughout and became progressively more listless. The conjunctivæ were injected.

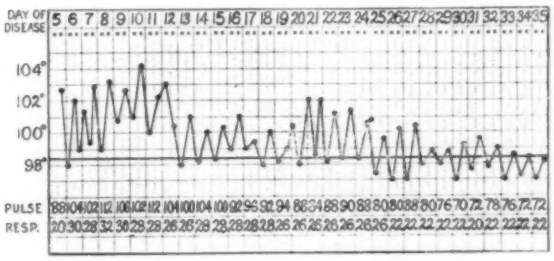


FIGURE I.
Temperature chart of J.O.

On the morning of the thirteenth day of illness (which was the ninth day in hospital) the temperature fell temporarily to normal. Although the fever continued for another nineteen days, it was never so high as in the first period, and the morning temperature thereafter was always normal or almost normal.

In the second week in hospital, œdema appeared in the ankles and fluid in the abdominal cavity. The sweating ceased. In the following week the patient became very ill. Signs of heart failure developed, and he appeared to become jaundiced. As the fever gradually fell the general condition improved slowly, and after the temperature became normal improvement was more rapid. All symptoms then disappeared, save deafness, which had begun soon after his admission to hospital and persisted throughout.

After the initial lobar pneumonia, treatment was symptomatic, except that sulphathiazole was given during the third week. He had no rash at any time, nor any palpable enlargement of lymph glands, liver or spleen.

The patient was discharged, convalescent, on October 9. Recovery of health was complete, though slow.

### Laboratory Tests.

Blood was collected on September 18 (the twenty-sixth day of illness) and submitted to the health department laboratory for agglutination tests. *Rickettsia burneti* was agglutinated to a significant titre (Table I) and *Proteus OXK* slightly (Table II). There was no agglutination with the other test organisms.

TABLE I.
*Results of Serum Agglutination Tests with Rickettsia Burneti.*

| Titre. | 26th Day. | 55th Day. |
|---|---|---|
| 1 : 10 .. .. .. | + + + | + + + |
| 1 : 30 .. .. .. | + | + + + |
| 1 : 100 .. .. .. | − | + + + |
| 1 : 300 .. .. .. | | + + + |
| 1 : 1,000 .. .. .. | | + |

A second sample of blood was collected on October 17 (the fifty-fifth day from the beginning of the illness). The titre of agglutination with *Rickettsia burneti* had increased considerably. The agglutination with *Proteus OXK* had disappeared.

TABLE II.
*Results of Serum Agglutination Tests with Proteus OXK.*

| Titre. | 26th Day. | 55th Day. |
|---|---|---|
| 1 : 20 .. .. .. | + + + | − |
| 1 : 40 .. .. .. | + + | − |
| 1 : 80 .. .. .. | + | − |
| 1 : 160 .. .. .. | − | − |

### Comments.

The great increase in titre of serum agglutination of *Rickettsia burneti* between the twenty-sixth and fifty-fifth days of illness establishes the diagnosis of Q fever. The slight agglutination with *Proteus OXK* is not regarded as diagnostic of scrub typhus. Such agglutination is occasionally found in cases in which scrub typhus can be excluded. There was no rash, eschar or enlargement of lymph glands to support a diagnosis of scrub typhus.

The temperature chart (Figure I) is of a type which is seen occasionally in Q fever. The majority of Q fever patients have a moderately high fever, continuous or somewhat remittent, lasting for six to fourteen days; the temperature then becomes normal and remains so. In some cases, however, the temperature falls at the usual time; but then, after a variable interval, there follows a relapse in which the rise in temperature is intermittent. The interpretation of J.O.'s chart is that it represents a primary period of fever lasting for twelve days, followed immediately by a relapse lasting for nineteen days.

No examinations for the presence of Q fever in the fauna of North Queensland have been made by us. Heaslip,[1] however, noted the presence in North Queensland of the bandicoot, *Isoodon torosus*, and the bandicoot tick, *Hæmaphysalis humerosa*, which provide respectively a reservoir and vector of Q fever in South Queensland. Bandicoots abound around Ingham and cattle are numerous.

One of the unsolved problems in the epidemiology of Q fever is the establishing of a link between the widely separated endemic centres of South Queensland and Montana, United States of America. The present case reduces the gap by a mere 500 miles. It is suggested that there may be a connecting chain of endemic centres along the eastern coast of Asia and adjacent islands.

### Summary.

A case of Q fever is described occurring at Ingham, North Queensland. This is the first proof of the presence of Q fever in North Queensland, and its occurrence extends the known endemic area by 500 miles.

### Acknowledgement.

One of us (E.H.D.) is indebted to Dr. J. Coffey, Deputy Director of Health and Medical Services, Queensland, for permission to publish this paper.

### Reference.

[1] W. G. Heaslip: "Tsutsugamushi Fever in North Queensland, Australia", THE MEDICAL JOURNAL OF AUSTRALIA, March 29, 1941, page 380.

## Reviews.

### "LETTSOM AND HIS TIMES."

IT is just a little more than a century since the classic biography of Dr. John Coakley Lettsom was published by his friend and colleague, Thomas Joseph Pettigrew; and it remained the standard authority on the life and work of the enthusiastic Quaker physician until Dr. James Johnston Abraham produced a revised version in 1933.[1] It would seem that Pettigrew's view of this remarkable personality was altogether too close to the scene; and in consideration of the fact that the new biographer had the good fortune to

[1] "Lettsom: His Life, Times, Friends and Descendants", by James Johnston Abraham; 1933. London: William Heinemann Medical Books Limited. 9¾" × 7¼", pp. 518, with many illustrations. Price (Australian): 23s. 9d.

unearth a great deal of fresh material, which revealed certain important incidents in a clearer light, he felt justified in telling the story all over again. That it has been told in the true spirit of literary craftmanship and historical research will be obvious to the reader who has applied himself to a thoughtful study of these fascinating pages.

It is difficult for a cold and calculating reviewer to resist the temptation of allowing himself to become excessively verbose and expansive in describing a book of this character, it paints such a vivid and colourful picture of Georgian England—of those boisterous days when so much of our present heritage of wealth, war and territorial possession was already being ruthlessly fostered and preserved. A long sequence of stirring events was crowded into the period of Lettsom's lifetime, many of them bearing a close analogy to momentous happenings within present recollection.

In order to preserve her integrity and further her ambitions, Britain found it necessary to be constantly at grips with the French and the Spaniards both on land and at sea. When Wolfe stormed the Heights of Abraham and Clive fought the battle of Plassey, Canada and India added lustre to the far-flung Empire overseas. It was not long, however, before a serious domestic error of judgement precipitated the American War of Independence; and then came the shock of the French Revolution to disturb the complacency and conservatism of all peace-loving citizens both at home and abroad. Finally, just as Lettsom was about to make a graceful exit from this mortal coil, Napoleon Bonaparte made his last bid for world supremacy, which ended in the illusion of a glorious victory at Waterloo.

During the latter half of the eighteenth century many great inventions and new ideas were beginning to transform England from an agricultural into a manufacturing country, and the progress of the industrial revolution was responsible for the growth of large cities, the accumulation of unprecedented wealth and the widespread prevalence of poverty, crime and disease. In the midst of these gross social inequalities religious fervour had reached sublime heights, and never before had the people been so strict in the outward observances of their faith. But Lettsom came to this England with a strangely unconventional background. In the first place he was a real "Colonial", having spent the early years of his life on a plantation in the West Indies; and then, all his thoughts and actions were tempered by the rigid discipline and simplicity demanded from every member of the Society of Friends. Throughout his career he remained loyal to the tenets of his religion and held steadfastly to its high ideals, so that he never hesitated to leave his fashionable consulting practice whenever his services were required in the nearby slums, in the disease-infested gaols or at one of the dispensaries, which he had inaugurated in the city of London for the treatment of the sick poor.

As we follow the varied activities of this indefatigable physician, there are many interesting sidelights on the medical institutions of the day. Here and there mention is made of the old system of apprenticeship and the conditions pertaining thereto. Frequent reference to the deplorable state of medical education throughout the country is consistent with evidence that the College of Physicians and the Corporation of Surgeons were completely indifferent to the unsatisfactory conditions under which medical students obtained their clinical experience, and to the fact that hundreds of incompetent surgeons and apothecaries carried on lucrative practices without let or hindrance with the passive approval of an uncomplaining public.

The author has gone to a lot of trouble to provide us with full information relating to the many public and philanthropic enterprises in the organization of which Lettsom was always a prominent figure. He was the founder of the Royal Society of London and nursed it through the early vicissitudes which threatened to imperil its very existence. He was an innovator in his conception of the idea to establish an open-air sanatorium for the treatment of the scrofulous, and through his efforts in 1796 the sick poor from London were able to benefit from the Royal Sea-Bathing Hospital at Margate. He was closely associated with the beginnings of the Royal Humane Society in 1774; and the description of some of the methods recommended by the society for the resuscitation of the apparently drowned is not without its humorous touches—if driving tobacco smoke up into the patient's large intestine by means of a tube introduced through the anus can be considered as such!

One of the outstanding features in the course of this biography is the lucid account of that important development which can be traced through the whole of the eighteenth century, and it originated from a world-wide movement to find ways and means to prevent the dreaded ravages of smallpox. The story begins with the efforts of Lady Mary Montague to introduce to her own country the method of inoculation which she had seen practised by a group of old women while she was a resident in Constantinople. Then came the adoption of arm to arm inoculation by the enterprising surgeon, Danial Sutton, but his system of prolonged isolation made the treatment possible only for people in comfortable circumstances, and this brought Lettsom into a heated controversy with Baron Dimsdale, because he thought it was high time that the method should be introduced into the London dispensaries for the benefit of the poor. Unfortunately the logic of Lettsom's arguments, in favour of mass inoculation was not as convincing as the persuasive eloquence of his literary style and he came out of the fray somewhat ingloriously. But some years later his active participation in the fight to establish the efficacy of Jennerian vaccination and to defend Edward Jenner's claim to priority in making the discovery, was an undertaking productive of more fruitful consequences.

The value of the book is considerably enhanced by the many excellent portraits and pictures which accompany the text. In it Dr. Abraham has given a delightful portrayal of the character of John Coakley Lettsom and a rare glimpse of the entrancing setting in which he lived and worked for the public weal.

## TUBERCULOSIS HANDBOOKS FOR DOCTOR AND PATIENT.

"PULMONARY TUBERCULOSIS", by R. Y. Keers and B. G. Rigden, is a handbook intended for students and practitioners.[1] It could perhaps be likened to a plate of good Scotch oatmeal, not exciting to the eye or palate, but containing much plain, wholesome nutriment in a small compass. The teaching upon bacteriology, pathology, epidemiology and immunology is up to date and orthodox and the book can be recommended to the medical library. In a foreword by F. H. Young (which digresses to become a reproof of one of the evils of State-controlled medicine) it is stated that "the emphasis of the book is rightly laid upon the treatment of the tuberculous patient as opposed to the tuberculous lung". However, this emphasis is not laid by any means as clearly as one would expect from the authors, who are no doubt in daily contact with their patients as the medical superintendent of a well-known Aberdeenshire sanatorium and his first assistant. The aim of treatment is put down as the complete healing of disease and the conversion of the patient into an "economic unit"; nothing, however, is said of the practical side of preparing the patient to enjoy life as best he can when *restitutio ad integrum* physically and economically is unlikely or impossible. The emphasis is on "firm discipline", and the authors strongly favour residence at a sanatorium wherever possible and prolonged bed rest. The reproductions from skiagrams are very good, and the book mark, a skiagram of a normal chest, for comparison with the illustrations in the text is ingenious.

"A Guide for the Tuberculous Patient", by G. S. Erwin, is well printed, attractively bound in paper boards, small enough to go in the pocket and very moderate in price.[2] The author is medical superintendent of a sanatorium in Cheshire and the text is reminiscent of an experienced sanatorium doctor's talks on tuberculosis to gatherings of his patients, satisfying their curiosity in simple language, seeking to gain their intelligent cooperation in treatment, but not imparting more information than it is good and useful for them to possess. "The tears of discipline are the waters of healing", the quotation at the head of the section on treatment, will no doubt be thought rather grim by sanatorium doctors in this country. Without taking Dr. Erwin too literally, it is fair comment and makes an apophthegm which is truer than his quotation to rejoin that the shedding of tears and any measure of recovery from tuberculosis never take place simultaneously. It is curious that in the important section on lowered resistance only the causes of physical strain are considered, while unhappiness and other kinds of nervous strain as a cause of lowered resistance are not mentioned. Still, the little book is full of ripe wisdom succinctly expressed.

[1] "Pulmonary Tuberculosis: A Handbook for Students and Practitioners", by R. Y. Keers, M.C. (Edin.), F.R.F.P.S. (Glas.), and B. G. Rigden, M.R.C.S. (Eng.), L.R.C.P. (Lond.), with a Foreword by F. H. Young, O.B.E., M.D. (Camb.), F.R.C.P. (Lond.), D.P.H.; 1945. Edinburgh: E. and S. Livingstone Limited. 7¼" × 5", pp. 290, with 125 illustrations. Price: 17s. 6d. net.

[2] "A Guide for the Tuberculous Patient", by G. S. Erwin, M.D.; 1944. London: William Heinemann Medical Books Ltd. 6¼" × 4", pp. 124. Price: 3s. 6d. net.

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## REEMPLOYMENT AND THE DISABLED.

AUSTRALIA has no greater internal problem than that connected with the reemployment in civil occupations of men and women engaged in active service with the several branches of His Majesty's Forces after their discharge. Everyone who has given any thought to the subject knows that it is not merely a matter of finding a job of work for an individual and of introducing him or her to it. Even those who were engaged in certain industries or occupations before they enlisted, whether they were merely beginners at the work in question or had achieved recognition as competent workers, may have developed to such an extent as to be unfitted for their previous type of work. Many were quite youthful when they became sailors or soldiers or airmen, and they had lived in a restricted environment and their mental outlook was just as narrow. But whether they were young at the outset or not, they have been partakers in world-shaking events in diverse and distant lands and have had as comrades men of other countries and other nations, different perhaps in manners, in culture and in temperament. It would be absurd to suppose that in the face of these complete and sometimes kaleidoscopic changes, men would in every instance be either willing or able to return to a former placid way of life. Every citizen has some share in the finding of ways and means to solve the problem of post-war civilian reemployment. Some people appear to think that a solution will be found by acts of Parliament that will declare that certain things shall happen in a particular way and that other things shall not happen in another. Legislation is necessary and can have certain results, but for success to be achieved in a matter of this kind the people at large must not only approve and understand, but must cooperate in every possible way. Personnel discharged from the services include many who suffer from disability occasioned by wounds or illness, as well as those who are physically and mentally whole. The disability may be permanent. Some place has to be found in post-war industry (the term being used in its widest sense) for all the disabled who are capable of earning a living and of making a contribution to the general welfare of society. No scheme of post-war rehabilitation can possibly be regarded as complete that does not make provision for them. With people of this kind, it goes without saying, medical practitioners must be actively concerned.

If a short statement was asked for on the factors to be considered when an attempt is made to find employment for discharged service personnel, those mentioned would include the physical ability of the person concerned, his mental equipment or status, his experience, attainments and skill in industry and his wishes for the future. To these should also be added the question whether, all these factors being considered, he will be likely to give satisfaction to his employer. This, of course, implies some appraisement of the employer as well as of the proposed employee. There are doubtless many who will be quite ready to find places in industry for service personnel who are discharged as fit, but will look askance at a man who returns to civil life having been wounded with perhaps the loss of part of a limb or of an eye or having suffered from some serious illness that has left him handicapped for the rest of his life. Some medical practitioners may be among their number. As a matter of fact, when disabled men are placed in industry in accordance with their abilities and their disabilities they give a good account of themselves. This fact, which should be more widely known, has been demonstrated in an extensive study made by V. K. Harvey and E. P. Luongo, medical director and assistant medical director respectively of the United States Civil Service Commission.[1] They investigated the accident experience, production, efficiency, sick absenteeism and turnover among 2,858 impaired workers in government industry and 5,523 able-bodied workers who served as controls. In the selection of impaired workers for study only those with serious physical defects were considered, defects most likely to be encountered in the placing of "disabled veterans", such as orthopædic defects (including amputations and deformities), visual and hearing defects, arrested tuberculosis and cardiac conditions. The able-bodied workers were selected at random from workers engaged in the same pursuits as those who suffered from disability, and were of the same sex and of approximately the same age, had had the same length of experience on the job and received the same salary. The workers were employed in 43 establishments of the War and Navy Departments and included those in craftsman, labourer, professional and service occupations. With the findings in regard to the occurrence of accidents we need not concern ourselves very much. The increased frequency rate for accidents among the physically impaired has something to do with accident proneness, which was found to be present more frequently among the physically impaired persons. Accident proneness enters into the placement of ex-service personnel in industry only in so far as it can be detected before the type of employment for the ex-service member is chosen. Harvey and Luongo find that it can to some extent be detected by means of certain psychological tests. One of the main findings recorded by Harvey and Luongo is that the productivity, both in quantity and quality, and the efficiency ratings of the physically impaired were found to compare favourably

[1] *The Journal of the American Medical Association*, April 7, 1945.

with those of the able-bodied. Unfortunately the percentage comparison figures cannot be given on account of lack of space. It must suffice to state that in the highest grades the figures were slightly in favour of the able-bodied, but in the next grades, comprising the great majority of both groups of workers, the figures were for all practical purposes equal. The highest rates for sick absenteeism were found among the administrative, professional, technical and scientific workers. It is thought possible that fatigue from predominantly mental stress may be a larger factor in short-term absenteeism than fatigue from predominantly manual or physical stress. Another finding was that a much lower rate of "turnover" (changing from one job to another) was found among the physically impaired than among the able-bodied, this "indicating that the impaired are superior from the standpoint of employment stability". This is not surprising after all, because once a physically disabled person is placed in a suitable job, he will be loath to leave it for fear he does not easily find another. Of the 2,858 impaired workers studied, 19·7% required changes in the job or "job tailoring" on placement. This low percentage is thought possibly to be an expression of the pressure of war work and of the urgency of getting persons on the job with a minimum of delay. In this connexion it is noteworthy that 90·3% of all impaired workers received no special considerations at all with regard to transportation, luncheon or rest periods, hours of work, methods of remuneration, special equipment or reporting time on clock or by card.

This study is a useful example to Australia. It shows what can be done in the way of investigations into efficiency in industry. It also shows that the United States authorities are eager to meet the needs of physically disabled persons—between October, 1942, and December, 1944, more than 44,000 persons with severe physical defects (they are shown in a table) were placed in government industrial establishments. To find suitable employment for disabled ex-service personnel will be more difficult than to find work for healthy persons. To achieve this end several requirements must be met. The first is that the ex-service man must be anxious and willing to work. Many of them are, but it is unfortunately true that much of the talk, political and non-political, that is current about service personnel, particularly those who have been disabled, is more likely to make them dwell on their disabilities than to help them to recover their confidence and their health. A self-reliant man, able to take an active part in industry, does not want a pension. There will always be a few who will be willing to do anything but work, and they are to be pitied. These statements are not to be looked on as reflecting in any way on the man suffering from a pensionable disability who is unable to work. The second requirement is that the disabled ex-service man shall receive all the assistance that medical examiners, departmental officers and other organized bodies can give him in his search for a suitable post; in fact much of the searching ought to be done for him. With this goes the requirement that employers should be willing to receive disabled men—they will do so if they are made cognizant of such investigations as those of Harvey and Luongo. Another requirement is that any difficulties in regard to compensation or pension in the event of a breakdown while at work shall be met by the introduction of suitable legislation. Finally, over and above all there is the never-to-be-forgotten fact that the sailor, soldier or airman who is discharged with a disability nearly always has a psychological as well as a physical problem. For this he may need treatment, and if he does, he will need to receive it individually at the hands of a trained psychiatrist. Nothing less will do.

# Current Comment.

## DYSMENORRHŒA.

DYSMENORRHŒA is a condition which is frequently discussed in medical literature. Of its clinical importance there is no doubt, and the divergent views put forward to explain it are a measure of our ignorance of its true nature. It has been discussed in these columns on several occasions, both as a condition affecting women generally and as having a bearing on the efficiency of women in industry. The most recent lengthy discussion appeared in November, 1942, when the observations of Helen M. Taylor on primary dysmenorrhœa were brought to the notice of readers. Another study of dysmenorrhœa comes now from two workers at the Women's Hospital, Liverpool, T. N. A. Jeffcoate and Sylvia Lerer.[1] The title of their contribution is "Hypoplasia of the Uterus with Special Reference to Spasmodic Dysmenorrhœa". That hypoplasia of the uterus is responsible for dysmenorrhœa is only one of the ætiological explanations that are offered. The theories of ætiology that have been advanced centre round mechanical obstruction, hypoplasia, psychogenic factors, constitutional factors and endocrine factors. There is no doubt that the theory of hypoplasia gained wide acceptance, but the psychogenic factors are recognized as of great importance. With the advances that have taken place in the sphere of endocrinology, sound reasons have been put forward and good clinical results reported to support the theory giving an all-important place in the causation to endocrine factors. There is no reason to suppose that any one theory of causation will ever be found to cover all cases of dysmenorrhœa, and it is possible to build up an explanation, incorporating factors of hypoplasia, endocrine origin and psychic origin that will explain most manifestations of the condition. When some of the conclusions of the enthusiastic and careful investigators of this subject are considered, it must be clear that what is lacking is basic knowledge of the physiological and biochemical behaviour of the tissue concerned and of its abnormal manifestations. When this is supplied all the rest will fall into place. In the meantime it is well to bear in mind the distinction that has to be made between the mechanism by which the dysmenorrhœal pain is produced and the underlying cause. Jeffcoate and Lerer summarize the views of many authors on the mechanism by which the pain is produced. In spasmodic dysmenorrhœa the pain is intimately related to the contraction of the uterine muscle, but the difference between painful and painless contraction is one of type rather than of strength. Increased muscle tone or tetany may occur, but there is much to be said for the theory which supposes incoordinate action or loss of uterine polarity to be the basic difference. A *corpus luteum* effect on the muscle is probably essential to painful contraction, but it is not yet known whether the effect is a positive one or a withdrawal phenomenon. It is also possible that muscle activity and hormones as well operate to some extent by inducing a state of ischæmia.

Jeffcoate and Lerer in their discussion on hypoplasia refer to the development of the uterus. They point out that it has been customary for clinicians to describe undeveloped uteri as being infantile, prepubescent or pubescent. They summarize what is known of the development of the uterus to show that the infantile state persists from birth to the prepuberty age. After this the uterus either remains infantile or assumes the adult state and

[1] *The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1945.

there is no evidence to support the existance of subdivisions. Theoretically there may be some place for a group in which the uterus is fully developed anatomically but is still incapable of full reproductive function, but in practice it would be difficult, if not impossible, to recognize this class. Three conditions are necessary for full uterine development. First of all the uterus must be endowed with an ability to utilize and to respond to ovarian hormones. Secondly, these agents must be produced and be available in sufficient amounts. Thirdly, an efficient circulation to assure an adequate supply of œstrogens to the uterus must be present. Hypoplasia may result if any of these factors are absent and it is not necessarily the result of defective ovarian function. Hypoplasia not infrequently results because the uterus is incapable of response by reason of some inherent fault. This explains why some patients with no other sign of endocrine disturbance have hypoplastic uteri and why the administration of œstrogens sometimes has no effect on the size of the uterus or on symptoms such as amenorrhœa. Hypoplasia, Jeffcoate and Lerer think, might also result from the presence of some factor having the power to destroy or to render inactive the œstrogenic hormone. These observers have analysed the histories of 829 patients suffering from spasmodic dysmenorrhœa. Only 27 of these had definite signs of hypoplasia. Certain patients had some minor malformation or malposition of the uterus, but these faults were not accepted as an indication of hypoplasia. Jeffcoate and Lerer point out that their total of 27 differs considerably from the estimates of some authors who hold that the incidence of hypoplasia is as high as 20% to 100%. They mention two authors whose estimates were 10%. They have confirmed the existence in a large percentage of cases of an interval between the occurrence of the menarche and the onset of dysmenorrhœa and advance this as evidence against the hypoplasia theory. They refer to another series of 86 patients each of whom had a hypoplastic or atrophic uterus; of 28 who were menstruating only one had dysmenorrhœa which was incapacitating. In regard to the fact that the effect of œstrogen on a hypoplastic uterus depends on the ability of the uterus to respond, they point out that if the uterus is insensitive, then œstrogen therapy is useless. They argue that if the uterus is sensitive, then there is no point in increasing its size temporarily unless the cause is corrected—the cause is usually underactivity of the ovary. And if the cause is successfully treated, then the hypoplasia is automatically cured and the administration of œstrogen is unnecessary. If œstrogen therapy does give relief it does not necessarily do so by overcoming hypoplasia.

Jeffcoate and Lerer's work, which is worthy of detailed study, is best interpreted as a plea for elucidation of the pathology of uterine hypoplasia. Even if this did not fill in all the gaps in our knowledge, it would probably make possible the more intelligent and more effective use of œstrogens.

## NUTRITIONAL AND OTHER CONDITIONS IN FRANCE.

COORDINATED information about the state of the people in a European country recently freed from Nazi domination is at present rather hard to come by. For this reason, if for no other, an article by C. Bettelheim,[1] Director of the Social Investigation and Research Centre of the Ministry of Labour and Social Security in the Provisional Government of France, will be welcome. Bettelheim deals with France since the liberation, and begins by pointing out the economic and social difficulties that have had to be overcome. The economic situation was affected chiefly by (*a*) the tremendous levies made on the French economic system during the period of occupation, (*b*) the destruction resulting from the war, and (*c*) the economic stagnation from which France had been suffering for many years before the war. The cost of the German occupation must have exceeded a million million francs; this does not take into account the systematic destruction carried out by the Germans, or the German requisitioning of goods. From the point of view of destruction resulting from the war, the most serious damage was done to the transport system; immediately before and immediately after liberation, the economic life of the country was completely paralysed because of lack of communications. A contributing factor to this paralysis was the shortage of coal and raw materials, which in turn was aggravated by the disruption of transport facilities. This situation inevitably reacted on industrial production, which in 1944 was only about 60% of the 1943 figure and 30% of the 1938 figure. Another factor to be taken into account was the absence in Germany of 2,000,000 Frenchmen—1,000,000 as prisoners of war, 60,000 as workers and 550,000 as political prisoners. The expansion of the French war effort up to the time of Germany's defeat must also be taken into account. A decline in labour productivity has also been observed; this is considered to be due to (*a*) the increasingly defective technical conditions of work due to worn-out machinery and to interruption caused by delays in the arrival of raw materials or the supply of electrical energy, and (*b*) the considerable decline in the standard of living, especially food supplies, clothing and housing.

Turning to nutrition, Bettelheim makes the following statement:

> It has been estimated that at the end of 1944 official rations provided about 1,255 calories [*sic*] per person per day, which is only 51·4 per cent. of the estimated normal ration of about 2,450 calories. This lack of nourishment is all the more serious because it has been accompanied by a decline in quality. In particular, the proportion of fats in the food ration has declined considerably, and at the end of 1944 only about 27·7 per cent. of the requirements in this field could be met. Here again the difficulty is explained by the fact, which is often ignored, that imports provided a large proportion of the food supplies of the country. This was particularly true in the case of fats, the consumption of which before the war was 47·8 ounces a month, of which about half was imported. At present the basic ration is only 8·8 ounces as a result of the falling-off in agricultural production.

The decline in agricultural production has affected the areas under cultivation for wheat, oats and potatoes, the amount of wine, meat and vegetables produced and the livestock position, which is serious. Causative factors have been shortage of labour, lack of equipment, lack of fuel and the inadequate supply of fertilizers, particularly nitrates and phosphates. The serious livestock position has been produced partly by the war and partly by lack of fodder; the drought of 1944 did nothing to help matters. In addition, the animals are in a permanent state of undernourishment. "The scarcity of fodder and its bad quality—due mainly to the shortage of phosphates in the ground—has affected the bone structure of young animals and reduced the output of milk, which is only a small fraction of the 1938 figure." The housing position is also serious. Before the war France was suffering from a scarcity of houses; the war has resulted in the destruction of about 1,500,000 dwellings. The problem of reconstruction is complicated also by the presence of approximately 100,000,000 mines in the ground. Bettelheim also discusses the financial situation in France, and brief mention may be made here of a few relevant points. The first is the existence during the occupation period of a considerable budgetary deficit, brought about by German levies. This could be only partly covered by loans, and the result was an enormous increase in the public debt. The decline in industrial and agricultural production, together with the considerable increase in the circulating currency, led to a rise in prices—about 150%. This rise had serious consequences for persons with fixed incomes and for wage-earners, whose wages did not keep pace with it.

The task of the Provisional Government of France has been no easy one. It will be impossible to discuss in detail in this place the measures it has taken to relieve the various aspects of the situation, although in general it may be said that the Government has had to cope with

[1] *International Labour Review*, June, 1945.

emergencies as they arose, and pursue a policy of expediency directed as far as possible by its already determined aims for the future. Repair work for the restoration of the transport system has been put in hand; reorganization of the general economic system has been begun, an important step being the dissolution of the Peasant Corporation (instituted by the Vichy Government) and the introduction of the industrial organization offices (*offices professionels*). Coalfields in two *départements* and the Renault factories have been nationalized. Prices have been kept down as far as possible by a system of subsidies; but sometimes this has been unsuccessful. The Government's social policy may be said to envisage the gradual raising of wages and of family allowances, and the reorganization of the system of old-age allowances for workers and of old-age and invalid pensions. This is necessary owing to the precarious position into which the social insurance funds have fallen as a result of actions taken during the Vichy *régime*. A further measure has been the policy of temporary displacement of workers to another locality, the workers agreeing to the transfer being given compensation according to the zone in which they are employed and to their family responsibilities. Workers who refuse to be transferred immediately lose their right to unemployment benefit. No worker is to be compelled to remain away from his home for more than six months. Finally, the Government has restored freedom of association and abolished Vichy's Labour Charter, instituted on October 4, 1941. The introduction of a system of works committees, to act as advisory bodies, has been the result of the programme drawn up by those working with General de Gaulle in London and also of a spontaneous movement in a number of factories immediately after liberation.

This in brief is the social and economic picture in France since liberation. It will not be out of place to draw attention to a review of the nutritional state of children in France by Harold C. Stuart,[1] Associate Professor of Child Health of Harvard University, Head of the Department of Maternal and Child Health of the School of Public Health, Boston, and recently medical adviser to the American Red Cross in France. From internal evidence in Stuart's article we gather that he spent some time in detention by the Germans. He begins by referring to the conflicting reports that have been received about the nutritional status of people in countries occupied by the Germans, and points out that whether one is favourably or unfavourably impressed by the appearance of the children in a foreign country depends (*a*) upon whether the observer had been familiar with these countries before the war, and (*b*) upon whether he is competent to appraise the nutritional status of children, a difficult undertaking even under optimal conditions. However, discrepancies between reports from different sections of France or between reports from France and neighbouring countries may be due to actual differences in local food supplies and health conditions. Rural areas in France have probably suffered far less than the larger French cities, and French children have probably suffered less than Belgian children. Stuart believes that it is possible to obtain a fairly adequate picture of the state of children in 1941, because careful studies carried out during that period have already been published. Enough material is also available from studies carried out in 1942 and the first half of 1943 to indicate the rate and extent of deterioration during that period. Stuart bases his review chiefly upon data from sources with which he himself is familiar: (i) studies conducted by the *Institut de Recherches d'Hygiène*, established in Marseilles early in 1941 with the assistance of the Rockefeller Foundation Health Commission; (ii) studies conducted in unoccupied France for the American Red Cross late in 1942. Discussing first the caloric intake, Stuart states that consideration of official rations is not satisfactory, because full rations frequently have been unobtainable, and the supplemented sources of Calories from unrationed foods have always been a considerable, though variable, factor. In 1941 Youmans, working in Marseilles, found the caloric value of the diet of children to be about 22% below the estimated requirements for age. The average intake for children of all ages was a little below 2,000 Calories per day. Kuhlman, continuing the same studies, reported for late 1941 an average consumption of 1,600 Calories. The studies in Marseilles were so thorough that it seems that reports of lower figures from other parts of France may have overlooked certain accessory sources of Calories. In the spring of 1943, Kuhlman, repeating the same studies, stated that children aged six to thirteen years were consuming on the average 1,725 Calories. This would make the average caloric deficiency in their diet about the same as that reported by Youmans. With regard to protein intake, Stuart considers that the evidence indicates that during the last three years the total protein provided by French diets has been close to the amount recommended, so far as children are concerned. However, only 19 grammes of the total of 63 grammes were obtained from animal sources, the remainder being obtained from vegetables. It is agreed that two-thirds of the protein in children's diets should come from animal sources, and that if this proportion is reduced, the total protein intake should be increased. This ratio has been reversed in the French diet. The lack of adequate Calories has probably brought about the burning up of much of the available protein for the production of energy, so that the lack of protein for structural purposes has been one of the major deficiencies in the French diet. However, strangely enough, œdema was never found, and when serum albumin determinations were made, hypoproteinæmia was not revealed. The children's growth and development were retarded, and they were "under par". Adequate or nearly adequate amounts of minerals were provided by the French diet. Calcium intake has been a little lower than the recommended amount, because of the lack of milk in the diet; but calcium has been obtained from other sources to a surprising degree. Radiological investigation failed to reveal calcium deficiency in more than a few of the older children, but evidence of rickets was commonly found in the films of infants. Iron intake has been adequate; but in spite of this fact, a considerable number of anæmic children have been found. The chief deficiency seems to have been in the number of erythrocytes. It may be that inadequate protein intake over a long period has been responsible. The only seriously deficient vitamin was vitamin *D*. Signs of early rickets were found in the X-ray films of all children examined who were aged between six and eighteen months. Deformities of healed rickets were often observed among older children, which must have been present before the war. The use of accessory sources of vitamin *D* in the feeding of infants seems never to have been a preventive measure adopted in Marseilles and in many parts of France. The finding of a high incidence of rickets among infants is not entirely an effect of the war. Children with florid rickets were often seen in the hospitals of Marseilles; but the incidence did not appear to have increased during the war. Vitamin *A* intake was also insufficient, but no clear-cut cases of active deficiency were found. Only a small percentage of children were found to have poor visual acuity in the dark, but the vitamin *A* level in the blood of 69% was below normal. It seems that some malnutrition may have resulted from the lack of true vitamin *A*, owing to the diet's poverty in butter fat and to the lack of reinforcement of butter substitutes. The position with regard to vitamin *B* was as follows: the provision of thiamin was adequate, that of niacin moderately deficient, that of riboflavin probably very insufficient. Although citrus fruits were unobtainable during most of the three past years, the ascorbic acid intake seems to have been adequate from potatoes and other sources. No evidence of deficiency of this vitamin was found. With regard to the general physical status and evidence of lack of growth and development among French children, Stuart states that casual observation showed that many were "under par"; they appeared listless and fatigued, were of poor posture and had small, flabby, relaxed muscles and poor development. Their stature for age was very short in comparision with that of American children. They were also slightly narrower in chest and pelvis, and their skeletal muscles were poorly developed. They tended to be lacking in subcutaneous tissue and thus to be underweight; but the average weight for age and height was very little below

[1] *American Journal of Public Health and the Nation's Health*, April, 1945.

the standards of Baldwin and Woods. Retardation in osseous age averaged 8·6 months for boys and 4·7 months for girls. From observations made in Lyons, Clermont-Ferrand, Montpellier and Monaco, Stuart concluded, in 1942, that "a state of general moderate to marked undernutrition was widespread in unoccupied France in the fall of 1942, at least among children over 10 years of age. There was little evidence that this undernutrition had generally reached a stage in which health had been profoundly affected, life endangered, or development more than moderately retarded". With regard to morbidity and mortality rates, Stuart believes that on the available evidence it is safe to conclude that no startling changes have taken place, although the rates have probably risen for the country as a whole. Stuart finally draws attention to a phenomenon which is little considered, though its effects may be profound—the powers of adaptation of the human body. In spite of almost universally poor diets, investigation of children in Marseilles as late as the spring of 1943 failed to reveal evidence of malnutrition in almost 50%. Stuart believes it probable that the surprisingly good condition of the children in France, after such a long period of malnutrition, may be due in part to this little-recognized capacity for adaptation. He concludes his review as follows:

> It is especially important to remember also that the adverse effects of war upon the children of Europe have not been confined to undernutrition and that full rehabilitation will require attention to many aspects of physical and mental hygiene. Crowding and unsanitary living conditions, lack of adequate public health control of communicable diseases and medical care of the sick have probably had at least as much to do with any increases in mortality rates as have deficiencies in diet.

## ELECTROENCEPHALOGRAPHY OF MIGRAINE SCOTOMATA.

MIGRAINE, always an interesting subject for inquiry, has taken on a new significance in that it is, with its curious scotomata, a frequent complication of decompression sickness. For years the belief has been growing that the visual disturbances are the result of spasm of the arteriolar branches of the posterior cerebral artery, whereas the headache arises from the nociceptive receptors in the extracranial branches of the carotid artery which are activated by dilatation. The homonymous character of the visual field defects, the sparing of central vision, the peripheral spread of the scotomata and the relief given by vasodilator drugs favour such an hypothesis.[1] It was inevitable that the new technique of encephalography should be directed towards this problem. F. A. Gibbs obtained definite evidence of reduction of electric activity in the contralateral occipital cortex in decompression migraine with scintillating scotomata; in brief the potential waves became slower and less regular when contrasted with the electroencephalogram from the ipsilateral occipital cortex.[2] When the visual field cleared the two records became identical. A recent encephalographic investigation of clinical migraine has revealed identical features. G. L. Engel, E. B. Ferris and J. Romano, of Cincinnati, examined three persons who were suffering from spontaneous migraine with scintillating scotomata (it may be noted that in their paper the word scotoma is frequently used as a plural!) and found unmistakable parallelism between visual disturbance and departure from normal of the record obtained from the contralateral occipital area.[3] As in the decompression cases, the slow and irregular waves of potential became quicker and more regular when the scotomata vanished, though the headache persisted. The visual manifestations of the clinical type can also be best explained by assuming a transient cortical ischæmia resulting from vasospastic action. So far so good, but no pathologist or physiologist has ventured to suggest a possible cause of the origin of the dazzling "fortifications" so often observed subjectively. The entering and retreating angles of these figures are acute, and, most remarkable of all, the lines bounding these angles, though restless, are straight. Nowhere in the body, in any cerebral or extracerebral structure, can a physical counterpart to these geometric figures be found. The surmise may be tentatively put forward that each straight line is the path of a vibrating particle, but just what sort of particle is not easy to predicate.

[1] G. A. Schumacher and H. G. Wolff: "Experimental Studies on Headache", *Archives of Neurology and Psychiatry*, Volume XLIV, 1941, page 199.
[2] *Transactions of the American Neurological Association*, Volume LXX, 1944, page 60.
[3] *The American Journal of the Medical Sciences*, May, 1945.

## UMBILICAL SEPSIS AND ACUTE INTERSTITIAL HEPATITIS.

THAT the umbilicus of the newborn child is still regarded by some pædiatricians as an important portal of entry for generalized sepsis is a statement with which J. Edgar Morison, of Queen's University, Belfast, begins a report on umbilical sepsis and acute interstitial hepatitis.[1] Though cases in which gross sepsis is obvious are generally recognized, some authors have shown that severe lesions may be present when external examination does not reveal much in the way of abnormality. Morison quotes Porak and Durante as having stated that, apart from such serious conditions as erysipelas, the more pronounced the umbilical lesion, the less severe is the general infection likely to be. Morison reports nine cases which should give rise to serious reflection in the minds of all practitioners engaged in obstetrics. In these nine cases, all of which came to autopsy, fairly uniform lesions of the liver were present together with sepsis "sufficient to delay the healing of the umbilical site, but of itself rarely sufficient to cause any clinical anxiety". The infants in these nine cases died between the ninth and the twenty-eighth days after birth. All except two of them were full-time infants and in only one case was a preeclamptic toxæmia present. In every case there was a constant history of an abnormal fall in birth weight with no recovery after the fourth day. In all but two cases diarrhœa, sometimes with vomiting, had become manifest by the seventh or eighth day. Only in two cases, however, could the severity, the continuance or the recurrence of the diarrhœa be regarded as significant. The diarrhœa did not respond to sulphaguanidine; and in three cases it may have been related to the time of development of a septic pulmonary complication. In three cases jaundice was in excess of any normal neonatal icterus. In two cases the umbilical cord had not separated, and in the other cases the umbilical site, which was usually invaginated, was still unhealed and there was a little dried blood or yellowish grey pus on the surface. Neither surrounding cellulitis nor localized peritonitis was present. The lesions in the liver were distinctive—nothing similar was found in 110 livers from infants in a strictly comparable age group or in another group of newborn or premature infants in which a less detailed survey was made. The essential lesion was an accumulation of mature polymorphonuclear leucocytes and relatively few mononuclear leucocytes in the portal tracts, especially in relation to the portal veins. These infiltrations were not related to the bile ducts and frank abscesses were not found. In six of the nine cases areas of pulmonary consolidation were present. They were multiple, discrete rather than diffuse, and extremely hæmorrhagic. They often presented a necrotic or purulent centre. Morison holds that the lesions were caused by infection, during the first few days of life, of the blood clot normally occupying the distal end of the umbilical vein in the body wall. He examines other possibilities, such as acute hepatitis of unknown origin and a primary intestinal infection, only to reject them. His conclusion must be accepted. He is obviously writing for the pathologist, but as already stated, the value of this paper to the obstetrician is considerable. Morison concludes, *inter alia*, that the aseptic technique of modern midwifery may sometimes fail. It is for the obstetrician to see that it does not.

[1] *The Journal of Pathology and Bacteriology*, October, 1944.

# Abstracts from Medical Literature.

## RADIOLOGY.

### The Radiographic Diagnosis of the Small Central Protruded Intervertebral Disk.

BENJAMIN COPLEMAN (*American Journal of Roentgenology*, September, 1944) states that there is a small group of patients with disk protrusions in whose myelograms the characteristic lateral defect produced by the usual disk protrusions is not found. In this group of patients the protruded portion of the disk is small and it lies centrally. The author stresses the importance of allowing the opaque oil to flow very slowly along the spinal canal so that the margins of a small protrusion will be outlined before the bulk of the oil obscures it. The myelographic examination is greatly facilitated by the use of "Pantopaque", a new medium developed especially for this purpose. It is of low viscosity, unproductive of reactions, tends to remain homogeneous and is not excessively opaque.

### The Intrapulmonary Lymphatic Spread of Metastatic Cancer.

H. PETER MUELLER AND RONALD C. SNIFFEN (*American Journal of Roentgenology*, February, 1945) state that lymphatic spread of cancer throughout the lungs represents the end-result of a carcinoma which usually originates in a distant organ, but occasionally within the lung itself. In most instances both lungs are evenly involved, but when the condition arises from an intrapulmonary tumour, or occasionally under other conditions, the lymphatic spread may occur on one side only or predominantly on one side. Radiographs show a diffuse, string-like formation of increased density, which radiates from the hilum toward the periphery. The trabeculæ break up into a fine network, scattered through which are numerous miliary nodules that usually correspond to the point of intersection of the trabeculæ, or represent fine lymph vessels which are hit edge-on by the X rays. The design in most cases is more marked in the central and basal portions of the lung, but it extends into the periphery and also into the upper portions. Often slightly larger nodules, due to large metastatic masses, are scattered between the linear markings and the fine miliary nodules. In most instances enlargement of the hilar nodes is obvious on the radiograph. Occasionally a small amount of fluid may be present in the pleural cavities. Anatomically there are two lymphatic systems within the lungs, one the deep intrapulmonary system and the other a more superficial pleural system, which drains the pleura and the most peripheral portion of the lungs. The intrapulmonary system extends through the bronchial and perivascular tissue and drains into the hilar lymph nodes. The pleural system runs in the connective tissue of the pleura and the interlobar septa, and also drains into the hilar lymph nodes, or to the thoracic duct. A sparse anastomotic circulation exists between these two systems at the periphery of the lungs. It is predominantly involvement of the deeper intrapulmonary system which leads to the characteristic appearance of intrapulmonary lymphatic spread. The authors discuss the differential diagnoses of miliary tuberculosis, pulmonary œdema and congestion, sarcoid pneumonokoniosis, and bronchiectasis.

### The Radiological Features of Eosinophilic Infiltrations in the Lungs.

HERMAN HENNELL AND MARCY L. SUSSMAN (*Radiology*, April, 1945) describe the clinical and radiological features of eosinophilic infiltrations in the lungs (Löffler's pneumonia). The authors state that there is a definite allergic background in most, if not all, of the cases, which suggests an allergic basis for this condition. A multiplicity of allergenic factors may be responsible for it. The radiological findings in the chest can thus be attributed to an exudative reaction in the lungs and pleura, perhaps analogous in character to the weal formation observed in the skin. It is in this way that the homogeneous density of the scattered and oblique plate-like areas can best be explained. The eosinophilic infiltrations are of homogeneous density and varying size. They may be confluent, resulting in a patchy appearance, or, if they are extensively confluent, resembling lobar consolidation. Sometimes there is an appearance of radiation from the hilum, but usually there are associated confluent patches at the periphery. Complete resolution usually takes place, but a few linear strands may remain. In this form, the radiological appearance is not characteristic. Tuberculosis or suppurative bronchopneumonia may be simulated. Certain phases in the course of Boeck's sarcoid are also similar. Frequently during the progress of the disease, however, narrow, plate-like homogeneous areas of density are seen extending obliquely caudad and laterally. Often they are symmetrical in the two lungs. They also resolve completely. Whether they represent localized exudations in the lung or in the pleura is impossible to state, but they seem to be unique to this disease. There is no predilection for upper or lower lobes, but a fair degree of symmetry on the two sides is the rule. Evidences of atelectasis, calcification and cavity formation are entirely lacking.

### Acute Obstruction of the Colon.

JOSEPH LEVITIN AND HELEN B. WEYRAUCH (*American Journal of Roentgenology*, February, 1945) state that a differential diagnosis between a slowly growing mechanical obstruction of the colon and an obstruction due to a sudden twist of the bowel such as a volvulus can be made on a preliminary radiograph of the abdomen. The slowly growing obstruction of the bowel is usually an annular carcinoma of the sigmoid, non-fungating, cicatrizing in type. This part of the colon is narrow and muscular. With increasing obstruction it maintains its tone and size. On the contrary, the caecum and ascending colon, being of greater diameter and thinner of wall, accommodate themselves to the increasing back pressure by dilating. A radiograph of the abdomen with the patient in the supine or prone position will show a colon filled with gas, to the point of obstruction. The caecum and ascending colon are on the right side, they are dilated, lie in a vertical direction and may have redundant loops. The transverse colon crosses the abdomen, it may also have redundant loops and is also dilated. The descending colon is well seen on the left side lying in a vertical direction, either not distended or with relatively little distension. The interruption of the gas-filled bowel is seen to be sharp at the point of obstruction. No large gas-filled loops are seen rising out of the pelvis. When volvulus of the sigmoid occurs there is a sudden twisting of the sigmoid bowel loop and it becomes a closed loop obstruction in relation to the rest of the bowel. This closed loop rapidly becomes greatly distended with gas and is seen to rise out of the pelvis and occupy the middle of the abdomen. It may extend to the diaphragm. At the summit the loop makes a sharp hair-pin turn. If a torsion is complete, the fluid in the bowel will exceed the gas present. A strangulated gangrenous bowel may show no gaseous distension, either of the involved loop or of the bowel proximal to the volvulus.

### A Simple Fluoroscopic Method of Foreign Body Localization.

CLARENCE J. ZINTHEO, JUNIOR (*Radiology*, October, 1944), describes a method for foreign body localization requiring only a standard table fluoroscope and the ordinary parallel arm caliper used in most departments to measure patient thickness. It consists essentially of locating, with the movable blade of the caliper, a point alongside the patient which is in the same horizontal plane as the foreign body, and is based upon the fact that shadows of objects in the same plane are displaced to an equal extent when the fluoroscope is moved. Since the movable arm of the common caliper is a blade an inch wide, some point on it must be defined which can be readily recognized on the fluoroscope. This is easily done by taping on the blade a small piece of wire, such as a bent paper clip, so that a point projects a fraction of an inch beyond the end of the bottom edge of the blade. It is the tip of this wire that is to be placed in the same plane as the object to be localized. The localization is performed in two steps: (i) Place the tip on the caliper in the same transverse plane (perpendicular to the table top) as the object. (ii) Raise the caliper arm until it is in the correct horizontal plane (parallel to the table top), as shown by equal shift of the tip and object when the fluoroscope is moved. To do this, stand the caliper on the table alongside the patient, with the blade beneath the screen so that the tip can be seen when the fluoroscope is turned on. Because of limitations of space beside the patient on the table top, the caliper may have to be placed parallel to the patient, only the tip being used as a reference point, not the whole arm as a pointer. Move the caliper along beside the patient until the tip is in the same transverse plane as the foreign body; this can best be done by closing the shutters to a narrow transverse slit with the shadows of the object and tip just barely seen simultaneously. Then open the shutters about two-thirds and move the fluoroscope until the shadow of one of the shutter edges just touches the shadow of the object. While holding the caliper standing where it was set in the first step, raise the movable arm until the shadow of the tip just touches the same shutter edge. The tip is then in the same horizontal plane as the

object. The caliper can easily be read in the fluoroscopic room light, and the dimension indicated is the distance of the object above the table top. The distance from the table to the top surface of the patient can be measured directly with the caliper, and the difference is the depth sought. Necessary precautions are few and simple. The slit for transverse alignment should be as narrow as possible; final positioning should be done with a slit so narrow that a slight change in the shutter closes off the X-ray beam entirely. The caliper must be firmly held in position while the shutter is opened, the fluoroscope moved for the second alignment, and the arm raised to final position. In the making of the final alignment, it is better to shift the fluoroscope in such a direction that the shutter edge towards the anode end of the tube is used, since in that way the focal spot is foreshortened and the shadow of the shutter is sharp, whereas if the edge toward the filament end is used the shadow is blurred and the accuracy of results impaired. An accuracy of one centimetre in localizing the depth is easily achieved, and with reasonable care better than 0·5 centimetre is not incompatible with speed.

## PHYSICAL THERAPY.

### Radiotherapy in Duplay's Disease.

M. O. R. NOBRE AND R. R. DE ARAUJO CINTRA (*The American Journal of Roentgenology*, October, 1944) discuss the ætiology, diagnosis and treatment of Duplay's disease, more commonly known as *peritendinitis calcarea*. In their article they suggest that massage is contraindicated and that surgery should be reserved only for cases in which a fair trial had been given to all other forms of treatment. The beneficial effect of radiotherapy may be explained by the property of X rays to destroy innumerable cells in the seat of infection, liberating antibodies and lysins which destroy the focal infection. They use an average dose of 150*r* to 200*r* and six or more applications, with an interval of two or three days between treatments. With the suppression of pain, an early result of radiotherapy, normal mobility is restored and the patient can return to his usual activity sooner than from any other treatment. During the first twenty-four hours after the irradiation, the symptoms are sometimes aggravated, but the following twenty-four hours bring a great relief in the pain and limitation of movement.

### Dosage Rate in Radiotherapy.

IN a symposium on dosage in radiotherapy, F. Ellis (*British Journal of Radiology*, November, 1944) points out that practical radiotherapists are concerned with the significance of dosage rate in its application to technique, and also with the fundamental biological principles involved. It is important to inquire if there is a difference in biological effect corresponding to differences in dosage rate, and also if a difference does exist, whether it is possible to define the critical values of dosage rate above and below which the effects are different. The conclusions to be drawn from the work on gene mutations are that dosage rate does not influence the effect which is a direct effect on the chromosomes. The dosage rate problem arose chiefly because Coutard's results of treatment of carcinoma of the larynx were published some years ago, and the low dosage rate of about 4*r* per minute to the skin was considered essential. However, it is true Coutard himself had not been able to use a higher rate because of the limitations of his apparatus. The author is convinced that cancer of the larynx can be cured with high dosage rate. The advantages of rapid treatment from the point of view of setting up the patient are obvious. It is easy to hold for a few minutes a position which would be impossible for an hour. In one field the possibility of a high dosage rate associated with a penetrating beam has produced results not produced in any other way; this is shown by the successful treatment of rectal carcinoma by the million volt apparatus at Saint Bartholomew's Hospital, London. The advent of contact therapy appeared to make the wave-length and dosage rate problems of no importance, since in this case the dosage rate is extremely high. The author therefore suggests that the highest dosage rate compatible with clinical accuracy and a suitable depth dose curve for the tumour concerned should be the criteria determining future apparatus construction.

### Cyrotherapy in Dermatology.

S. M. BLUEFARB (*Archives of Physical Medicine*, May, 1945) points out that there are physical and chemical methods which will produce peeling of the skin. A third method, combining the two, is one in which a carbon dioxide slush is used—it is known as cyrotherapy. It is an adjuvant method in the treatment of certain dermatological conditions. An exfoliation of the corneous layer of the skin is produced; the process rarely extends beyond the corneous layer. The author describes the technique. Solid carbon dioxide is placed in a mortar and ground to a fine powder. Precipitated sulphur (3% to 5%) is added to the "snow". Pure acetone is poured into this mixture, while it is constantly stirred, until a "slush" is produced. This should have the consistency of soft water ice. The mixture is applied with a tampon of absorbent cotton covered with a square of gauze. A rolling motion and some pressure are used. The procedure is repeated three to six times at one sitting. At each application a temporary blanching of the skin occurs. Immediately after the treatment the skin becomes hyperæmic and in two or three days peeling will be noted. The resulting deposit of sulphur is allowed to remain on the skin for ten to fifteen minutes after the treatment. The author discusses the contraindications to the use of cyrotherapy.

### Muscle Spasm in Poliomyelitis.

A. L. WATKINS AND MARY A. B. BRAZIER (*Archives of Physical Medicine*, June, 1945) present a preliminary report on the results of electromyographic studies on the effect of various forms of thermal therapy and of prostigmine in muscle spasm due to poliomyelitis. The clinical material consisted of six adult patients in the early infectious stage of the disease. In all, 835 measurements were made, the first test usually being made within a few days after the establishment of the diagnosis. The forms of treatment tested were: (i) the Kenny type of hot packs, applied for twenty minutes; (ii) infra-red irradiation for twenty minutes; (iii) luminous heat, applied for twenty minutes; (iv) diathermy, applied for twenty minutes; (v) the intramuscular injection of 1·6 milligrammes of prostigmine. Muscle spasm was measured by quantitation of the electrical discharges released on five seconds of passive stretching by known weights. It was found that single applications of hot packs, infra-red irradiation and diathermy had no effect. The application of luminous heat and the injection of prostigmine caused a decrease in spasm of approximately 25%. This degree of change was not considered sufficient to indicate a specific effect on spasm. The patients felt that the different types of heat were all moderately comforting, but no consistent preference was expressed for any particular type. All disliked prostigmine because of its effect on the gastro-intestinal tract. For the first few days of the disease several patients had severe pain in the affected extremity. None of the methods of treatment investigated was outstandingly successful in relieving it, and from clinical observation the authors gained the impression that the pain tended to decrease spontaneously rather than as a result of treatment. The authors conclude their report with the statement that further studies on the repeated use of a single method of treatment will be necessary before it can be evaluated.

### Effects of Electrical Stimulation on Denervated Skeletal Muscle.

W. H. WEBRMACHER, J. D. THOMSON AND H. M. HINES (*Archives of Physical Therapy*, May, 1945) have studied the effects of electrical stimulation on denervated skeletal muscle. They used the gastrocnemius muscle of albino rats and bilateral denervation was produced by removal of a section of the tibial nerve. The denervated muscle of one limb served as the experimental member and that of the contralateral limb as its control. The muscles received volleys of electrical stimuli for periods varying from half a second up to 180 seconds. The treatments were given on alternate days, or once a day or twice a day. The treatments were begun on the day after section of the nerve and were discontinued on the day before the tests were made. Control experiments were carried out. The results indicated that the atrophy of muscle following denervation can be appreciably retarded by electrical stimulation. The optimal intensity was found to be of the order of that required to elicit maximal tension values on comparable exposed muscles. Weak stimuli proved to be ineffective. The treatments were most effective when they were applied under conditions allowing maximal physiological stretch. The effectiveness of the treatments was within wide limits independent of the length of the periods of stimulation and proportional to the frequency of the treatment periods. Little difference was noted between the effectiveness of different forms of electrical stimulation if they were capable of eliciting comparable amounts of tension in the muscles. The authors believe that their findings offer some suggestions for the selection of an effective plan for the treatment of muscles after peripheral nerve injury.

# British Medical Association News.

## SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on May 31, 1945, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. E. A. TIVEY, the President, in the chair.

### Blast Injuries.

SURGEON COMMANDER C. KEATING read a paper entitled "Immersion Blast Injuries of the Abdomen" (see page 207).

SURGEON CAPTAIN LAMBERT ROGERS read a paper entitled "Blast Injury of the Brain" (see page 209).

MAJOR R. H. B. BETTINGTON read a paper entitled "Injuries to the Ear due to Blast" (see page 210).

PROFESSOR C. G. LAMBIE said that those who had had experience of war injuries only during the first world war had everything to learn about blast injuries from those who had had experience of the different type of warfare in which they were common. It had been a great pleasure and privilege to have at the meeting men who had been able to observe these injuries at first hand. Such injuries did occur in the first world war, but only a few people commented on them—such men as Stokes, Logan and Mott. Most of these injuries were put down to psychological causes—"psychic shock"—and Cuthbert Wallace ("Saint Cuthbert") described an incident in which two soldiers had been blown up when they lit a fire over a spot under which lay an unexploded shell. The post-mortem examination revealed no injuries; Wallace said that the men died from psychic shock. Obviously they died from blast injuries. Professor Lambie went on to say that a friend of his, in London, reported an incident in which a flying bomb exploded 200 yards from his house; no injury was sustained in the house except a broken pane of glass, but 400 houses in the neighbourhood were completely destroyed. Hitler, too, had had an escape; it was on record that a bomb had exploded at a table under his feet, and he had miraculously escaped. Professor Lambie said that he had one question to ask; it concerned cerebral hæmorrhages resulting from blast. He asked Surgeon Captain Rogers what was his view of the mechanism by which these hæmorrhages were produced. It had been suggested that they were due to sudden compression of the thorax causing a wave of back pressure in the veins, which could not yield because they were in the unyielding skull. Professor Lambie was not satisfied with this explanation.

SURGEON COMMANDER H. R. I. WOLFE said that he thought that the problem of blast injuries had been adequately dealt with and he had little to add. It was most important, however, that a distinction should be made between blast produced by air and under-water blast. He had had experience mainly of under-water blast injuries, chiefly in the Mediterranean. At that time he was working in a hospital ship, and many patients were received from twelve to twenty-four hours after the infliction of their blast injuries. Some casualties were received earlier. Blast injuries fell into three clinical groups, though these fused. In the first there was profound shock, associated with rupture of the viscera and hæmorrhage. The damage was so great that little could be done for the patient. The second included those patients in whom a viscus had been ruptured at the time of injury. The patient arrived in hospital, shocked, complaining of severe pain and with a rigid and painful abdomen. There were signs which warranted laparotomy. Surgeon Commander Wolfe said that he was in complete agreement with what Surgeon Commander Keating had said about patients who had sustained under-water blast injuries of the thorax and not of the abdomen—relaxation could be observed if the examination was carefully carried out. The third group had sustained an under-water blast injury, but not of sufficient magnitude to produce perforation. The patients were shocked to a mild or moderate degree and presented signs of bowel damage, but no signs to warrant laparotomy. In these cases there were often physical and radiological signs of concomitant lung damage. Surgeon Commander Wolfe quoted the cases of two such patients who were admitted to the hospital ship in which he was working. Both did well, and then one on the sixth day and one on the seventh day complained of acute abdominal pain; peritonitis developed, and they died in twenty-four hours. In acute blast injuries the mortality rate was high; in delayed perforation following blast injuries the mortality rate was appalling. This fact might be due to the occurrence of injury to the caecum and the sigmoid colon at once. Commander Wolfe said that the only such injuries he had seen in this type of case were pin-point perforations in the gut and retroperitoneal hæmorrhages. If one was in doubt whether to operate or not, a very strict watch should be kept on the patient, and if he complained of severe and increasing abdominal pain, tending to be localized in the lower part of the abdomen, immediate laparotomy was indicated. If the operation turned out to be unnecessary recovery might be jeopardized; but the mortality rate from delayed perforation from such injuries was very high and warranted exploration. Referring to the use of sulphonamides in the abdomen, Commander Wolfe said that if the powder was put in as a fine hoar frost it was justifiable. However, he did not think that this war-time expedient should be adherred to too rigidly in elective peace-time surgery, because of the danger of adhesions. He had seen two patients who had had a stormy convalescence with plastic peritonitis three weeks after a "clean" exploratory operation. Commander Wolfe did not think the intraperitoneal use of sulphonamides in such cases was justified.

SURGEON COMMANDER ST. GEORGE DELISLE GRAY said that he had recently encountered several cases in which blast might have been a factor. During the last war, His Majesty's ship *Russell* was sunk outside Malta harbour. A day or two later several of the casualties were buried in Malta. Shortly after the funeral, several sudden deaths occurred among people who had attended it, including the padre. The deaths were put down to fumes or something which had been inhaled. In 1917 the *Osmaniyeh* was torpedoed outside Mlio, and about 500 people were in the water. Destroyers laid depth charges, which were fairly new in those days, and not many had been dropped. Next day several of the survivors from the *Osmaniyeh* died rather suddenly with symptoms of "abdominal shock". To turn to the present war, on a recent occasion a bomb exploded near a number of people working on the deck of a ship. One young officer was standing about twenty yards away from the bomb; he was knocked over. He sustained several small penetrating wounds around his scapulæ, but no other injury was visible; he was extremely ill and looked moribund. He was semiconscious and was simply given morphine and transfusion of serum and plasma. Twenty-four hours later he was much better. When examined about ten days prior to the meeting—a fortnight after he had looked as if about to die—he was perfectly fit. Three officers were standing on the bridge just near where the bomb went off, and they "ducked". They all had their ear drums perforated. One had the *membrana flaccida* torn, another had a U-shaped perforation round the lower end of the malleus, yet another had a long perforation running almost to the top of the drum in front of the malleus. Commander Gray said that he entirely agreed with what Major Bettington had said about syringing ears that had been damaged by blast. The effect of blast on the ear was just the same as a blow from another person's elbow or from a football on the side of the ear—the drum was torn. If the injury was left alone, recovery took place in time, and it was impossible to see that a perforation had been sustained. An article had been published some time earlier in the *British Medical Journal*, dealing with about 100 cases of perforation of the ear drum in the desert. All the injuries were treated with sulphonamide insufflations; about a third of the patients had chronic suppuration afterwards. Commander Gray then referred to another case of rupture of the ear drum; the subject was diving, wearing a diving helmet. He was about fifty feet below the surface, when he felt pain in his ear and heard a pinging noise. It was rare for a subject actually to hear the rupture of his ear drum occur, because usually there was so much noise going on outside. Referring to flying bombs and blast, Commander Gray said that a flying bomb had exploded about 100 yards in front of his flat, and all the glass in the front was blown in. A peculiar thing was that drawers that were closed at the time of the explosion, and also closed hat boxes, were later found to contain fragments of glass, although no holes could be seen through which the glass had entered. Glass was also found inside the piano which was closed at the time. Commander Gray also referred to an explosion that had occurred in a ship's hold; in compartments that had no direct connexion with the hold pieces of material from the hold were found.

DR. G. C. HALLIDAY said that he had been interested in the effects of blast on the ear when he was in the Middle East; he had spent some time with other ear, nose and throat surgeons in Cairo investigating these effects. At that time they were receiving at his hospital a great many casualties

from the desert. Dr. Halliday had been struck by the number of infections associated with these blast injuries; in the first batch of sixty of these casualties, the infection rate was something like 78% or 80%. The treatment at that stage was reasonably good—nothing was done at all, and sterile wool was put in the ear. They had found that when any syringing had been done, or even if spirit drops had been instilled, infection was almost always the end result. When a mastoid operation had to be performed, the infecting organism always proved to be a hæmolytic streptococcus. It was considered that in these cases it would be wise not to put sulphanilamide powder into the ear, so a sulphonamide was given by mouth. Over the period in which they were able to carry out this idea, they were under the impression that results were better. In one case, a man had in the desert received a blast injury of minor consequence. He remained on duty. One week later he was transferred to Alexandria on account of a septic throat condition. He complained of deafness, and was sent to another ear, nose and throat surgeon. The surgeon found a central perforation of the ear drum and instilled spirit drops. The man at once became giddy, vomited and fell on the floor. Within ten hours he had intense otorrhœa; later facial paresis developed, and finally he had to undergo a radical mastoid operation. Dr. Halliday remarked that this case emphasized the danger of any form of treatment in the presence of a central perforation. He was unable to agree with Commander Gray, who had said that these patients recovered; many did not. The outcome depended largely on the amount of infection present. Dr. Halliday had seen a man with a large kidney-shaped perforation; he had had no infection, but no one would have known that the perforation was not due to chronic infection. In civil life everybody should be informed that nothing should be put into the ear in cases of traumatic injury to the ear drum. Dr. Halliday then referred to prophylaxis and the avoidance of deafness. He said that he had been able to examine the entire artillery regiments of the Ninth Division; less than 3% had ear perforations, and 80% showed evidence of deafness. No specialized instruments were available to test the hearing; the tests had to be made with a watch and the whispered voice. Many of the men were retested twelve months later, and in a number of cases the deafness had persisted. Dr. Halliday had gone into the question fully; it was extraordinary how prophylaxis was attempted in the different batteries. He could not agree that cotton wool was of no use. These men used cotton wool, rubber plugs, *crêpe* rubber; some simply applied their fingers to the aural orifice. They were all sure that when they did not protect their ears they experienced pain and ringing, and they took care next time. In some batteries it was noticeable that some took a "tough" attitude; they attempted no protection, and deafness was much more common. Dr. Halliday was sure that a great deal of protection could be afforded to people in the artillery at least. He had seen three men who had been in a tank which was blown up in Syria. Two had ruptured drums in both ears; the third had one drum ruptured only, because in the other ear there was a hard plug of wax which had afforded protection.

DR. E. P. BLASHKI said that he had been interested in Major Bettington's remarks about the influence of blast on a patient who already had some degree of deafness such as was present in otosclerosis, and the subsequent increase. Dr. Blashki said that he had had the opportunity of seeing on the Pensions Appeal Assessment tribunal a number of men who had served in the last war. He was struck with the number of such men who suffered from otosclerosis, which appeared to have made unexpected and considerable progress because of their war service. The ears of any man serving in the war were undoubtedly exposed to some degree of blast injury; such men were all exposed to noise of some sort. Dr. Blashki had been impressed some years earlier with a case which appeared to be one of perfectly simple otosclerosis. The man had been given a pension on the recommendation of the late Sir James Barrett, who had said that although the condition seemed to be ordinary otosclerosis, it was impossible to be certain that it was not aggravated by the patient's war service. The decision had seemed too lenient at the time; but Dr. Blashki took a different view now. Deafness in such cases did appear to be accelerated and exacerbated by exposure to noise. Dr. Blashki had not seen so many patients with blast deafness as the men in the services; but in many cases blast injury seemed to leave a high degree of deafness. That was a point that needed to be borne in mind by service medical officers, and also by those who had to do with pensions; too much should not be discounted. Another question arose when a man at the present time, twenty-five years after the last war, claimed that his deafness, which was the ordinary deafness of advancing years, was due to his service in that war. It was a difficult matter; but in some cases the men should be given the benefit of the doubt.

SURGEON COMMANDER STEELE PERKINS referred to the use of the sulphonamide drugs on the skin and in particular in the ear. He said that there was an ever-increasing number of people who had been sensitized to sulphonamides. In one or two cases of rupture of the ear drum in which sulphonamide powder had been used, sulphonamide sensitization had been induced, so that the drug could not be used without a general reaction. Another point was that in the ear this powder formed a most annoying crust, sufficient to cause *otitis externa*. If infection was present, boric powder and iodine was the best application to use; it was less irritating than sulphanilamide powder, and could be removed with a sucker if necessary. The best treatment was no treatment and simply observation until some emergency arose.

SURGEON REAR ADMIRAL J. MAXWELL expressed his appreciation that members of the Royal Naval Services should have been asked to take part in the discussion, and said that he thought that they had constructed two interesting papers. Rear Admiral Maxwell then said that he agreed with Commander Wolfe that men with early blast injuries were less prone to peritonitis than men with delayed effects. Finally, Admiral Maxwell said that he could not forbear to enter the zoological catalogue. In the hospital ship in which he had served in the Mediterranean, some bombs were dropped close alongside. There were rats in the ship, real rats, inconspicuous and rare; but after the explosions for weeks the ship was alive with rats. The question was, were they disturbed, or were they seeking laparotomy?

Surgeon Captain Lambert Rogers, in reply to Professor Lambie's question, said that if Commander Gray would tell him how the glass got into the hat boxes and the closed drawers, he would be in a better position to tell Professor Lambie how the cerebral hæmorrhages took place. He also felt sceptical about Mott's explanation. What seemed to him to be more satisfactory was the suggestion that the backlash, the rebound wave, might be responsible for leakage from certain vessels. However, with Professor Lambie, he remained puzzled about the precise mechanism.

Dr. Halliday spoke again. He said that he had given up the local use of the sulphonamides in the outer ear; he had found it was much more harmful than useful. He thought that given by mouth it was successful. On several occasions he had stopped short with 25 or 30 grammes, and the ear condition flared up; a mastoid operation was performed, and he found only inflamed air cells, but no pus. After that experience he continued with sulphonamide treatment for a much longer time, and no more mastoid operations became necessary. He had not had to perform one in six months at the military hospital he was attending.

DR. TIVEY, from the chair, said that three interesting papers had been read and an interesting discussion had been heard. In the first place, he wished to convey, on behalf of the New South Wales Branch of the British Medical Association, thanks to Surgeon Rear Admiral Maxwell, and also to Surgeon Captain Lambert Rogers and Surgeon Commander C. Keating for their interesting papers. Dr. Tivey also thanked the other medical officers of the Royal Navy who had attended the meeting and contributed to the discussion. In the discussion of blast injuries, Dr. Tivey said that he had been rather surprised that thoracic injuries were not more emphasized. He had been under the impression that during the "blitz" a considerable number of deaths were due to pulmonary collapse and various other forms of lung injury. Dr. Tivey also thanked Major Bettington for the aspect of the case that he had presented from the point of view of the aural surgeon. It had been a great pleasure to have the speakers at the meeting, and the evening had been most instructive.

DR. A. J. COLLINS, in moving a vote of thanks, said that for those present who had served in the last war, the mysteries of blast did not exist. The nature of explosives had changed so much that blast, with which the modern medical officer had come into contact, was something quite new to the experience of the older members. It was interesting to them who had read of the phenomenon, but had had no contact with it, to hear what had been said at the meeting. They were thankful to have the Royal Navy medical personnel with them for many reasons. It was hoped that such meetings would in the future be more prominent and more prevalent. It was also hoped that before the present war was over similar programmes would be repeated. There were many aspects of war medicine and surgery with which the Royal Navy medical officers were more familiar than Australians. Once every six months

something should be attempted. Dr. Collins also expressed the hope that some of the naval medical officers would attend the ordinary meetings of the Branch.

DR. GEORGE BELL seconded the vote of thanks. He said that he had much enjoyed the meeting. As Dr. Collins had remarked, the difference in explosives no doubt accounted for the different experience of medical men in modern warfare compared with the experience of those in the last war. Dr. Bell had been interested in Surgeon Captain Lambert Rogers's remarks about injuries to the brain; there again modern explosives accounted for a vast difference in experience. Members of the Branch were particularly pleased to have the visitors with them and hoped that they would be present on future occasions.

The vote of thanks was then carried by acclamation.

# Post-Graduate Work.

## LECTURES AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that the following are the concluding lectures in the winter lecture series for civilian medical practitioners and service medical officers of our own, British and Allied services which will be held at the Stawell Memorial Hall, 145, Macquarie Street, Sydney:

*Monday, August 20, at 4.30 o'clock p.m.*—"Cineplastic Amputation", Lieutenant-Colonel B. K. Rank; "Observations on Plastic Surgery", Surgeon Lieutenant-Commander G. Humby, R.N.V.R.

*Tuesday, August 28, at 4.30 o'clock p.m.*—"The Chemotherapeutic Control of Malaria", Brigadier N. Hamilton Fairley.

Full information concerning these and other activities of the Post-Graduate Committee in Medicine may be obtained from the Secretary of the Committee, 131, Macquarie Street, Sydney. Telephone: B 4606.

## A SPECIAL COURSE IN GYNÆCOLOGY AND OBSTETRICS AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that, in conjunction with the honorary medical staff of the Royal Prince Alfred Hospital, a course in gynæcology and obstetrics will be held at the King George V Memorial Hospital for Mothers and Babies, Camperdown, from September 17 to September 28, 1945.

The fee for the course will be £5 5s.; £2 12s. 6d. per week will be charged for board and residence at the hospital. Registration for the course will be limited to twenty-four persons. Applications for registration, accompanied by a remittance for the amount of the fee and the amounts for board and residence, if required, should be made to the Secretary, New South Wales Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney.

The programme is as follows:

### Gynæcology.

*Monday, September 17.*

9.30 a.m.: Welcome by Dr. H. H. Schlink, Chairman of the Board of Directors. Address by Dr. S. A. Smith, Chairman of the New South Wales Post-Graduate Committee in Medicine. 11 a.m.: "Dysmenorrhœa", Dr. Muriel McIlrath. 2 p.m.: "Interpretation of Pain in Gynæcology", Dr. F. A. Maguire. 3.30 p.m.: "Fibroids: Variety, Symptoms and Treatment", Dr. M. B. Fraser. 8 p.m.: Film. "Treatment of Face Presentation."

*Tuesday, September 18.*

9.30 a.m.: Lecture Demonstration. "Malformations of the Genital Tract", "The Surgery of Prolapse", Dr. H. H. Schlink. 11 a.m.: Operative Demonstration. "Manchester", Dr. H. H. Schlink. 2 p.m.: "Bacteriology and Pathology of Post-Abortional Septicæmia", Dr. Mary Heseltine. 3.30 p.m.: Operative Demonstration. "Repair of Prolapse using Diathermy", Dr. F. A. Maguire. 8 p.m.: Film. "Caudal Anæsthesia."

*Wednesday, September 19.*

9.30 a.m.: "Pelvic Inflammation", Dr. C. Chapman. 11 a.m.: "Sterility", Dr. F. N. Chenhall. 2 p.m.: "Post-Abortional Sepsis", Dr. C. Chapman. 3.30 p.m.: Lecture Demonstration. "The Vulva at the Menopause", Dr. F. N. Chenhall. "Vulvo-vaginitis", Dr. Murial McIlrath. 8 p.m.: Film. "Eclampsia."

*Thursday, September 20.*

9.30 a.m.: "Bleeding at and after the Menopause", Dr. F. A. Maguire. 11 a.m.: Operative Demonstration. "Total Hysterectomy", Dr. F. A. Maguire. 2 p.m.: "Gynæcological Pathology", Dr. Mary Heseltine. 3.30 p.m.: "Anæsthesia in Gynæcology", Dr. W. I. T. Hotten.

*Friday, September 21.*

9.30 a.m.: "Early Diagnosis and Treatment of Uterine Carcinoma", Dr. H. H. Schlink and Dr. C. Chapman. 11 a.m.: Operative Demonstration, Dr. H. H. Schlink. 2 p.m.: Operative Demonstration. "Surgical Aspects of Uterine Carcinoma", Dr. C. Chapman. 3.30 p.m.: Lecture Demonstration. "Ovarian Cysts: Variety and Treatment", Dr. M. B. Fraser. "Menorrhagia", Dr. M. McA. White. 8 p.m.: Film. "Breech Presentation."

### Obstetrics.

*Monday, September 24.*

9.30 a.m.: "Forceps", Professor B. T. Mayes. 11 a.m.: "Pyrexia in the Puerperium", Professor B. T. Mayes and Dr. Mary Heseltine. 2 p.m.: "Medical Complications of Pregnancy", physicians and senior obstetricians. 4 p.m.: "Diseases Peculiar to the Newborn", Dr. N. C. Cunningham. 8 p.m.: Film. "Forceps."

*Tuesday, September 25.*

9.30 a.m.: "The Care of the Vagina and Perineum during and after Delivery", Dr. H. A. Ridler. 11 a.m.: "The Cervix in Pregnancy and Labour", Dr. H. A. Ridler. 2 p.m.: "Radiology in Obstetrics", Dr. D. G. Maitland. 3.30 p.m.: "Causation of Stillbirths and Neonatal Deaths", Dr. Mary Heseltine. 8 p.m.: Film. (i) "Episiotomy and Repair", (ii) "Fœtal Birth Injuries".

*Wednesday, September 26.*

9.30 a.m.: "Toxæmia of Pregnancy", Dr. J. Chesterman. 11 a.m.: Clinical Round, Dr. J. Chesterman. 2 p.m.: "Toxæmic Problems", Dr. R. F. Back. 3.30 p.m.: "Lower Segment Cæsarean Section", Professor B. T. Mayes. 8 p.m.: Film. "Lower Segment Cæsarean Section."

*Thursday, September 27.*

9.30 a.m.: "Prolonged and Delayed Labour", Dr. B. Williams. 11 a.m.: Clinical Round, Dr. B. Williams. 2 p.m. to 5 p.m.: "Demonstration of Selected Prenatal Cases", Professor B. T. Mayes.

*Friday, September 28.*

9.30 a.m.: "The Rh Factor: Its Relation to Babies", Dr. Kathleen Winning. 11 a.m.: "The Care of the Premature Baby", Dr. Kathleen Winning. 2 p.m.: "The Surgery of the Newborn", Dr. M. R. Flynn. 3.30 p.m.: "Obstetrical Pathology", Dr. Mary Heseltine.

On Monday, September 24, Professor Bruce Mayes takes the full class. On all other days the classes are halved.

One-half of the class will take gynæcology the first and obstetrics the second, and the times will be reversed for the second half of the class.

Motion films will be screened on certain evenings during the course. Attendance at these sessions is purely optional.

# Correspondence.

## THE PHARMACEUTICAL BENEFITS ACT, 1944.

SIR: A circular, dated 5th July, 1945, has been issued by the Western Australian Branch of the Association, requesting financial contributions to a Publicity Fund to fight the *Pharmaceutical Benefits Act* as at present constituted in its "plan of attack" (circular, 18th May, 1945).

At the foot of this first circular is a statement, thus: "If no reply is received it will be assumed that you are unwilling to contribute." We must assume, on our part, that this statement has some significance, or it would not have been put there. May it be asked: "What is the significance of it?"

One's alternatives are: (i) To send a contribution because one supports the Association's "attitude" to the act. (ii) To

apologize because one supports it, but cannot afford to contribute. (iii) To contribute because one does not wish to be henceforth labelled as one "unwilling" to contribute (that is, financially support the Association's "attitude"). (iv) Not to reply and be so labelled.

It should be reasonable to think that the statement in question was not prompted by idle curiosity as to which members of the Association do not support the Association's "attitude". Why then is the Association so keen to categorize its members so?

To rank and file members it may appear that the Association is trying to dragoon unwilling individuals into supporting its "attitude". It may also appear that the Association authorities are preparing a black list, that perhaps we are facing another "Fishbein purge".

The fear which some members have of opposing the Association's policy is very real, and was recently displayed to me when I wrote a letter to the lay Press in which I generally supported the act. I received a number of congratulatory messages from both laymen and members of the profession. One doctor wrote and told me that he thought I had been "courageous". Why courageous? Why is one supposed to be a hero merely because his opinion differs from that of his Association? Personally I cannot see it, and feel that unqualified statements such as the one on the circular referred to only help to make some feel that they must submit lamely to every dictum of the Association or run a very real and grim risk of the displeasure of the leadership of the Association.

It seems impossible to me that a state of terrorism and dictatorship could exist in a professional body, and I suggest that the air would be considerably cleared if the leaders of the Western Australian Branch gave the rank and file a clear explanation of the statement referred to, and an assurance that no dragooning or black-listing is contemplated, and that members are individually free to either support or oppose the legislation concerned.

Yours, etc.,

LESLIE R. JURY.

Big Bell,
Western Australia,
July 20, 1945.

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SIR: Replying to the above letter from one of our members—Dr. L. R. Jury, of Big Bell—we attach herewith a copy of the notice to which he refers, circulated to members on July 5 on the back of an agenda paper. We think this is self-explanatory.

Yours, etc.,

R. D. McKELLAR HALL,
Honorary Secretary, Western Australian Branch, British Medical Association.

Shell House,
205, St. George's Terrace,
Perth.
August 2, 1945.

[COPY.]

**Items of Interest to Members.**

*Publicity:*

The meeting of the Convocation of Groups' Representatives held on June 10 produced a strong and widespread demand for better and more active publicity for the Branch's attitude to the Pharmaceutical Benefits Act. The need to publicise our views will increase with every medico-political development. The Merredin Group has asked for the appointment of a paid lay director for our publicity. This idea had previously been considered by the Council's Publicity Committee and reluctantly dropped for lack of funds.

Publicity costs money—at least £1,000 p.a. for this State. Are you prepared to pay for this?

So long as the broadcasting of the Branch's opinion depends on the spare time and spare energy of already very busy medical practitioners, the amount and effectiveness of that publicity will be limited.

By filling in and returning the undertaking below you will assist the Branch Council to determine how much publicity will be possible.

S. E. CRAIG, President.

I am willing to contribute to the Publicity Fund of the West Australian Branch the sum of £..... (up to £5).

Signature .....

Address .....

If no reply is received it will be assumed that you are unwilling to contribute.

# Naval, Military and Air Force.

## APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 136 and 144, of July 12 and 26, 1945.

NAVAL FORCES OF THE COMMONWEALTH.

### Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

*Transfer to Emergency List et cetera.*—Surgeon Lieutenant (for Short Service) Athol Herbert Robertson is transferred to the Emergency List and reappointed for temporary service, dated 27th March, 1945.

### Citizen Naval Forces of the Commonwealth.

*Royal Australian Naval Reserve.*

*Promotion.*—Surgeon Lieutenant Alan John Gray is promoted to the rank of Surgeon Lieutenant-Commander, dated 1st March, 1945.

*Appointment.*—Alan Richard Whately is appointed Surgeon Lieutenant (D), dated 14th June, 1945.

*Termination of Appointment.*—The appointment of Alexander Francis Murray as Surgeon Lieutenant is terminated, dated 3rd May, 1945.

*Royal Australian Naval Volunteer Reserve.*

*Promotion.*—Surgeon Lieutenant Ernest Favenc Chin is promoted to the rank of Acting Surgeon Lieutenant-Commander, dated 31st March, 1945.

*Confirmation in Rank.*—Surgeon Lieutenant (on probation) Ernest Favenc Chin is confirmed in the rank of Surgeon Lieutenant, with seniority in rank of 31st March, 1941.

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THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 149, of August 2, 1945.

ROYAL AUSTRALIAN AIR FORCE.

*Citizen Air Force: Medical Branch.*

Temporary Flight Lieutenant O. W. Bowering (285170) is granted the acting rank of Squadron Leader whilst occupying a Squadron Leader post with effect from 23rd February, 1945.—(Ex. Min. No. 179—Approved 1st August, 1945.)

The appointment of Temporary Flight Lieutenant W. J. Shaw (252479) is terminated at his own request with effect from 4th May, 1945.

The following Flight Lieutenants are transferred from the Reserve to the Active Force with effect from the dates indicated: R. Roxburgh (267591), P. R. Bull (257476), R. K. Doig (257656), L. C. Dunlop (267739), T. Schlight (257727), J. R. Tripp (267568), L. C. Doubleday (267797), J. P. Walsh (257477), J. A. S. Robertson (267525), W. R. Pitney (257530), J. V. Hurley (257659), D. M. Clarke (257635), G. E. W. Bennett (267613), E. W. Bate (257611), 13th May, 1945, C. Lancaster (257597), F. J. Gray (267808), W. L. Morris (267522), E. R. W. Thomson (277232), W. F. A. Harris (297466), C. H. Walsh (267801), J. H. A. Floyd (257657), J. J. Nattress (257665), A. H. Toyne (267719), A. M. Beech (257693), V. T. Stephen (257670), R. A. Godfrey-Smith (257579), L. J. Weinholt (277543), C. I. Wilkinson (277479), 17th June, 1945.

The notification in respect of the appointment to a commission of Paul David Victor Moni (277528) appearing in *Commonwealth of Australia Gazette*, No. 27, dated 8th February, 1945, is cancelled.

The probationary appointments of the following Flight Lieutenants are confirmed with effect from 3rd June, 1945: W. J. Betts (287464), L. R. Trudinger (257718).

The grants of the acting rank of Squadron Leader to the following Temporary Flight Lieutenants are terminated upon their ceasing to occupy Squadron Leader posts with effect from the dates indicated: G. A. Hodgson (263612), 17th May, 1945, C. W. Kingston (273945), 18th May, 1945, H. G. Andrew (283733), 29th May, 1945.

The appointment of Temporary Squadron Leader S. D. Mecoles (253432) is terminated on medical grounds with effect from 21st May, 1945.

The appointment of Temporary Flight Lieutenant H. G. Andrew (283733) is terminated at his own request with effect from 1st June, 1945.

The appointments of the following officers are terminated on medical grounds with effect from the dates indicated: Temporary Squadron Leader J. S. Bothroyd (251461), 11th June, 1945, Temporary Flight Lieutenant R. H. Oxby-Donald (255253), 14th June, 1945.

*Reserve: Medical Branch.*

Temporary Wing Commander C. P. Hudson (261240) is transferred from the Active List with effect from 11th May, 1945.

Temporary Wing Commander C. P. Ley (261213) is transferred from the Active List with effect from 3rd May, 1945.

The following medical practitioners are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: Paul David Victor Moni (277528), 23rd November, 1944, Charles Edward Young (297490), 26th March, 1945, Lefteri Comino (277539), 30th March, 1945, George Corones (277538), 7th April, 1945, Maurice John Matthews (287466), 14th April, 1945, Benjamin Edward Brookman (287468), 21st May, 1945.—(Ex. Min. No. 185—Approved 1st August, 1945.)

The following officers are transferred from the Active List at their own request with effect from 24th May, 1945: (Temporary Squadron Leaders) K. H. Hill (251689), G. Mackintosh (262616).

Temporary Squadron Leader D. L. Peate (261262) is transferred from the Active List with effect from 7th June, 1945.

The following officers are transferred from the Active List with effect from 13th June, 1945: Temporary Wing Commander R. G. Plummer (281253), Temporary Squadron Leader L. T. Conlon (261679).

## Medico-Legal.

### HOCKING VERSUS BELL.

THE High Court of Australia on August 10, 1945, dismissed an appeal by Mrs. Stella Hocking, of Quirindi, New South Wales, against a ruling of the State Full Court in which the court entered a verdict for Dr. George Bell, with costs of all trials and appeals. The judgements in this case were published in THE MEDICAL JOURNAL OF AUSTRALIA of September 2, 1944, at page 248. The High Court verdict was by a three to two majority.

## Obituary.

### CHARLES BATTANDIER MACVEAN.

WE regret to announce the death of Dr. Charles Battandier MacVean, which occurred on July 9, 1945, at Parramatta, New South Wales.

### CHARLES RETCHFORD.

WE regret to announce the death of Dr. Charles Retchford, which occurred on July 30, 1945.

### ROBERT ALGERNON FOX.

WE regret to announce the death of Dr. Robert Algernon Fox, which occurred on August 8, 1945, at Darlinghurst, New South Wales.

## Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

D'Ammond, Phyllis Kathleen Annie, M.B., B.S., 1943 (Univ. Sydney), "Glencourse", Hazelbrook.

Cohen, Morris, M.B., B.S., 1944 (Univ. Sydney), 113, Station Street, Newtown

O'Keefe, Eva Florence, M.B., B.S., 1941 (Univ. Sydney), 86, Queenscliff Road, Manly.

## Diary for the Month.

AUG. 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 22.—Victorian Branch, B.M.A.: Council Meeting.
AUG. 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 24.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 28.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 30.—New South Wales Branch, B.M.A.: Branch Meeting.
SEPT. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 5.—Western Australian Branch, B.M.A.: Council Meeting.
SEPT. 5.—Victorian Branch, B.M.A.: Branch Meeting.
SEPT. 6.—New South Wales Branch, B.M.A.: Special Groups Committee.
SEPT. 6.—South Australian Branch, B.M.A.: Council Meeting.
SEPT. 7.—Queensland Branch, B.M.A.: Branch Meeting (Jackson Lecture).
SEPT. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 11.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
SEPT. 14.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 17.—Victorian Branch, B.M.A.: Hospital Subcommittee.
SEPT. 17.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
SEPT. 18.—Victorian Branch, B.M.A.: Organization Subcommittee.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

*Victorian Branch* (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract practice appointments in Western Australia. All Public Health Department appointments.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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